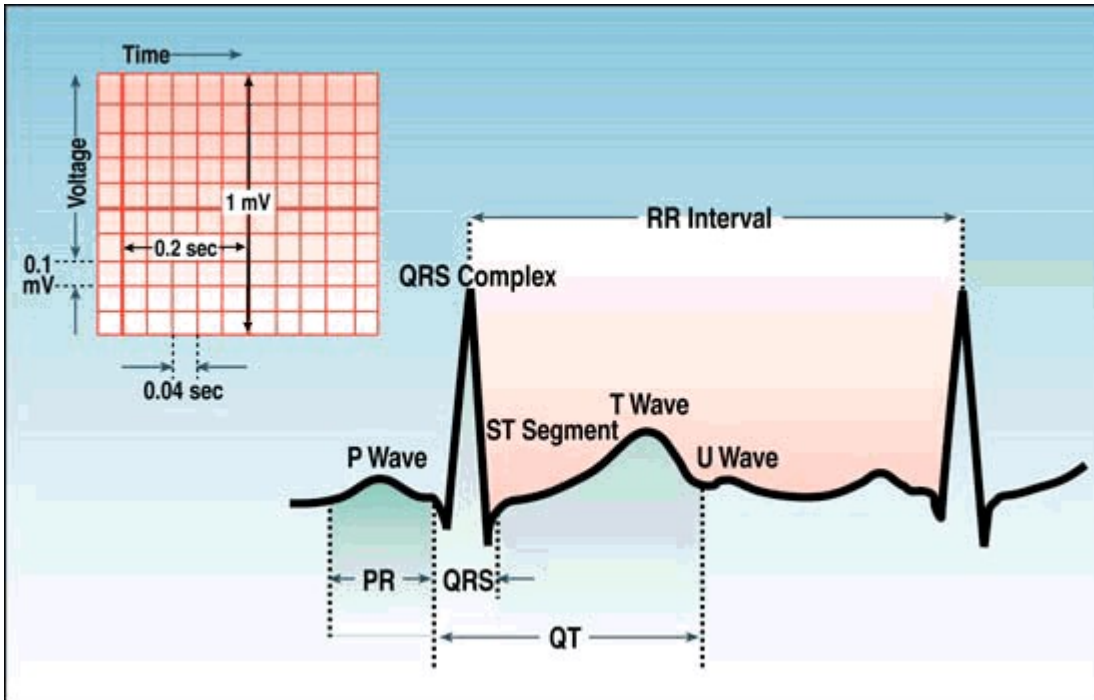


I. The Standard 12 Lead ECG

The standard 12-lead electrocardiogram is a representation of the heart's electrical activity recorded from electrodes on the body surface. This section describes the basic components of the ECG and the lead system used to record the ECG tracings.
Topics for study:

1. [ECG Waves and Intervals](#)
2. [Spatial Orientation of the 12 Lead ECG](#)



This diagram illustrates ECG waves and intervals as well as standard time and voltage measures on the ECG paper.

1. ECG Waves and Intervals:

What do they mean?

- P wave: the *sequential* activation (depolarization) of the right and left atria
- QRS complex: right and left ventricular depolarization (normally the ventricles are activated *simultaneously*)
- ST-T wave: ventricular repolarization
- U wave: origin for this wave is not clear - but probably represents "afterdepolarizations" in the ventricles
- PR interval: time interval from onset of atrial depolarization (P wave) to onset of ventricular depolarization (QRS complex)
- QRS duration: duration of ventricular muscle depolarization
- QT interval: duration of ventricular depolarization and repolarization
- RR interval: duration of ventricular cardiac cycle (an indicator of ventricular rate)
- PP interval: duration of atrial cycle (an indicator of atrial rate)

2. Orientation of the 12 Lead ECG

It is important to remember that the 12-lead ECG provides spatial information about the heart's electrical activity in 3 approximately orthogonal directions:

- Right ↔ Left
- Superior ↔ Inferior
- Anterior ↔ Posterior

Each of the 12 leads represents a particular orientation in space, as indicated below (RA = right arm; LA = left arm, LF = left foot):

➤ Bipolar limb leads (frontal plane):

- Lead I: RA (-) to LA (+) (Right Left, or lateral)
- Lead II: RA (-) to LF (+) (Superior Inferior)
- Lead III: LA (-) to LF (+) (Superior Inferior)

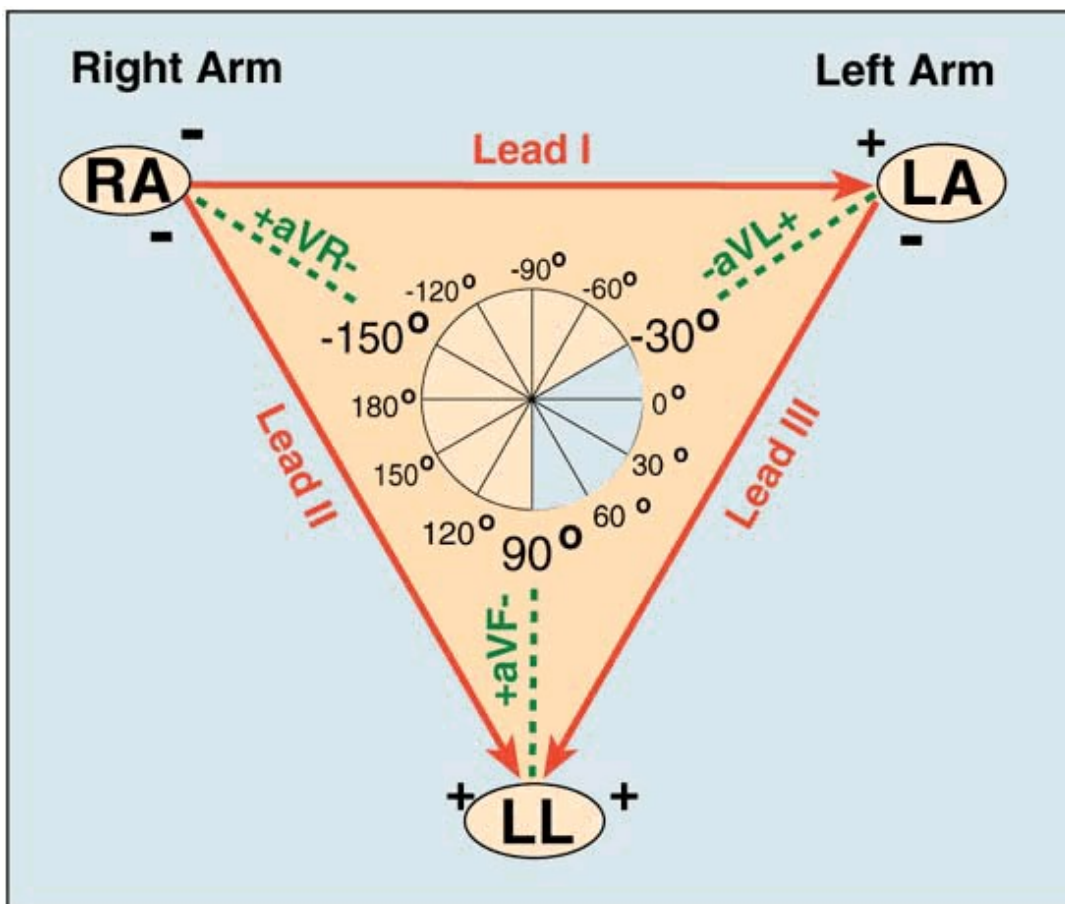
➤ Augmented unipolar limb leads (frontal plane):

- Lead aVR: RA (+) to [LA & LF] (-) (Rightward)
- Lead aVL: LA (+) to [RA & LF] (-) (Leftward)
- Lead aVF: LF (+) to [RA & LA] (-) (Inferior)

➤ Unipolar (+) chest leads (horizontal plane):

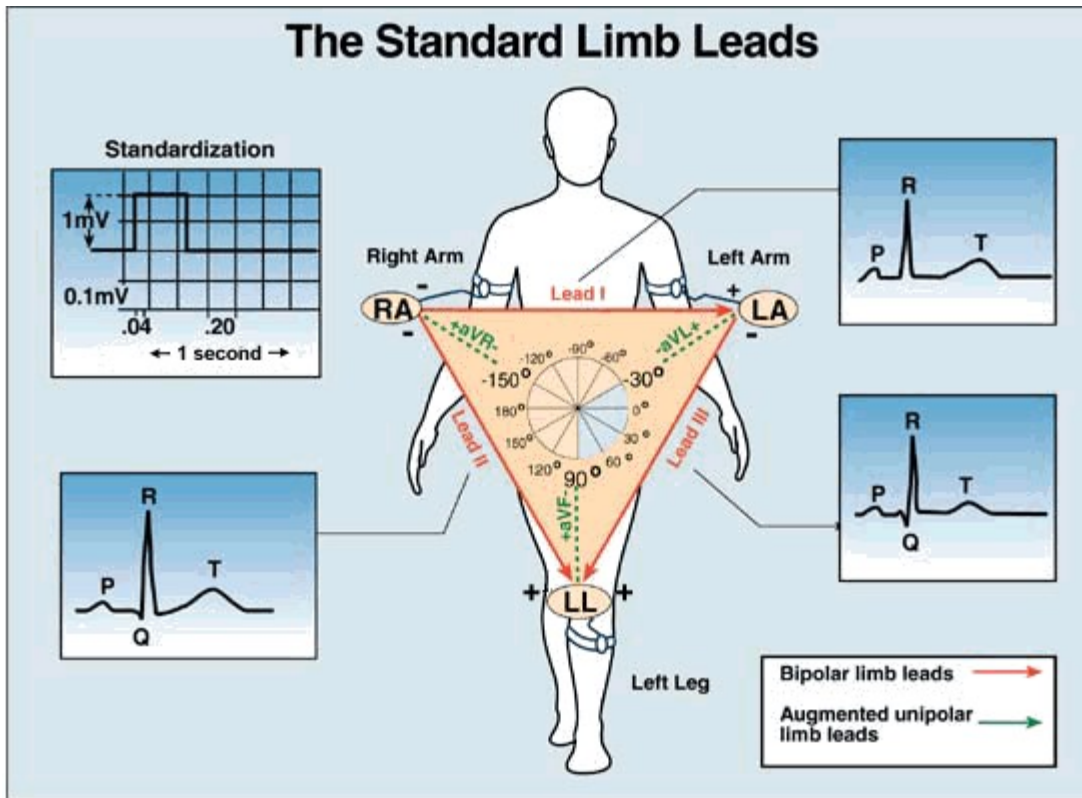
- Leads V1, V2, V3: (Posterior Anterior)
- Leads V4, V5, V6: (Right Left, or lateral)

Lead Placement Diagrams

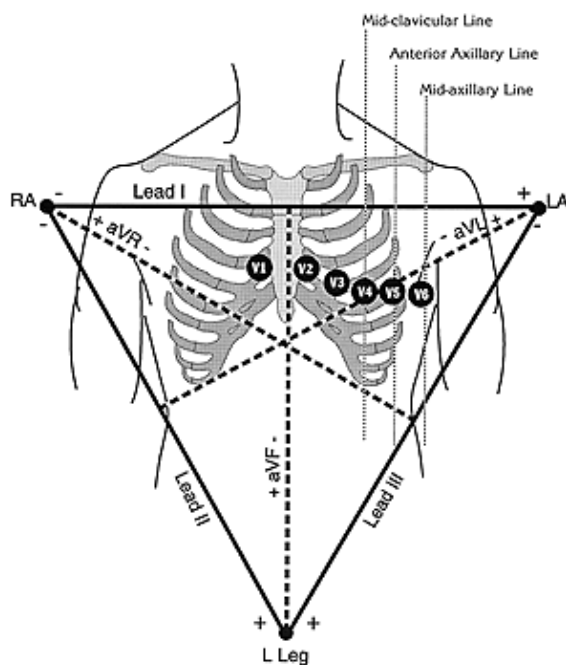


Behold: Einthoven's Triangle! Each of the 6 frontal plane leads has a negative and positive orientation (as indicated by the '+' and '-' signs). It is important to recognize that Lead I (and to a lesser extent Leads aVR and aVL) are right ⇌ left in orientation. Also, Lead aVF (and to a lesser extent Leads II and III) are superior ⇌ inferior in orientation. The diagram below further illustrates the frontal plane hookup.

The Standard Limb Leads



LOCATION OF CHEST ELECTRODES IN 4TH AND 5TH INTERCOSTAL SPACES:



V1: right 4th intercostal space

V2: left 4th intercostal space

V3: halfway between V2 and V4

V4: left 5th intercostal space, mid-clavicular line

V5: horizontal to V4, anterior axillary line

V6: horizontal to V5, mid-axillary line

II. A "Method" of ECG Interpretation

This "method" is recommended when reading all 12-lead ECG's. Like the physical examination, it is desirable to follow a standardized sequence of steps in order to avoid missing subtle abnormalities in the ECG tracing, some of which may have clinical importance. The 6 major sections in the "method" should be considered in the following order:

[Measurements](#)

[Rhythm Analysis](#)

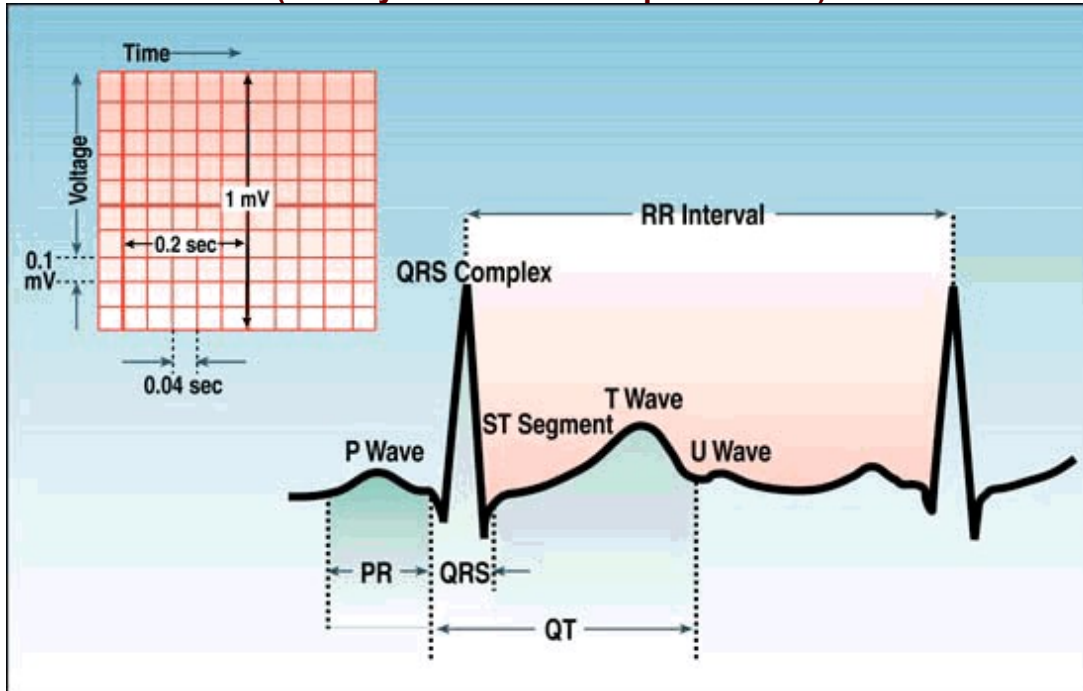
[Conduction Analysis](#)

[Waveform Description](#)

[Ecg Interpretation](#)

[Comparison with Previous ECG \(if any\)](#)

1. Measurements (usually made in frontal plane leads):



- Heart rate (state atrial and ventricular, if different)
- PR interval (from beginning of P to beginning of QRS)
- QRS duration (width of most representative QRS)
- QT interval (from beginning of QRS to end of T)
- QRS axis in frontal plane (go to: ["How To Determine Axis"](#))

Go to: [ECG Measurement Abnormalities \(Lesson IV\)](#) for description of normal and abnormal measurements

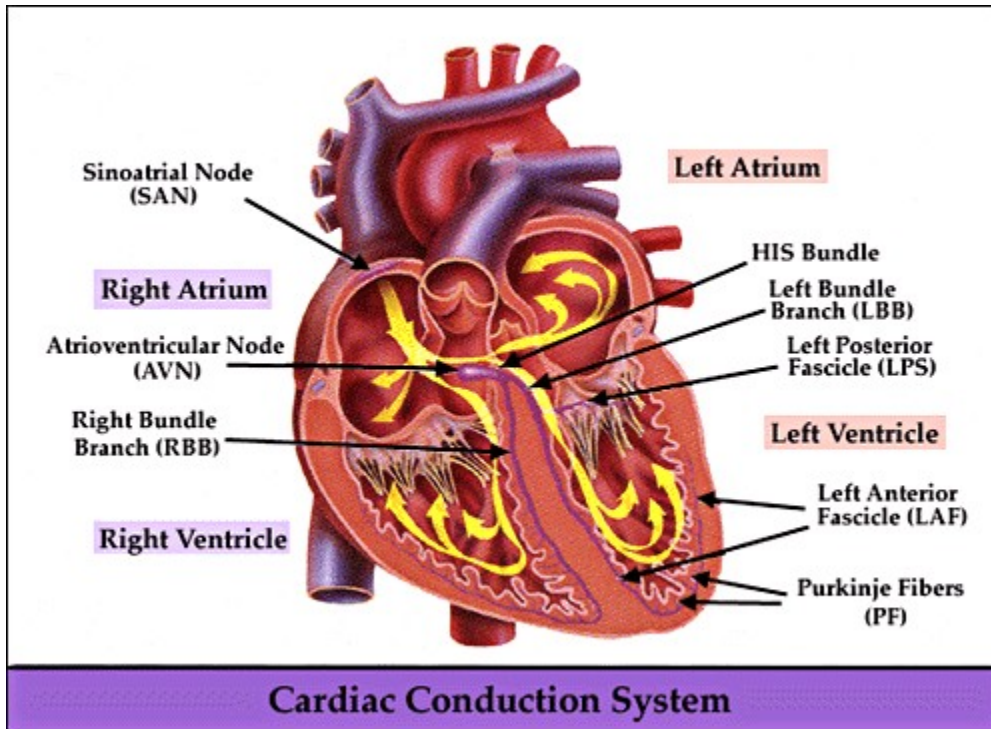
2. Rhythm Analysis

- State basic rhythm (e.g., "normal sinus rhythm", "atrial fibrillation", etc.)
- Identify additional rhythm events if present (e.g., "PVC's", "PAC's", etc)
- Consider all rhythm events from atria, AV junction, and ventricles

Go to: [ECG Rhythm Abnormalities \(Lesson V\)](#) for description of arrhythmias

3. Conduction Analysis

➤ "Normal" conduction implies normal sino-atrial (SA), atrio-ventricular (AV), and intraventricular (IV) conduction.



➤ The following conduction abnormalities are to be identified if present:

- [SA block \(lesson VI\)](#): 2nd degree (type I vs. type II)
- [AV block \(lesson VI\)](#): 1st, 2nd (type I vs. type II), and 3rd degree
- [IV blocks \(lesson VI\)](#): bundle branch, fascicular, and nonspecific blocks
- Exit blocks: blocks just distal to ectopic pacemaker site

(Go to ECG [Conduction Abnormalities \(Lesson VI\)](#) for a description of conduction abnormalities)

4. Waveform Description

➤ Carefully analyze the 12-lead ECG for abnormalities in each of the waveforms in the order in which they appear: P-waves, QRS complexes, ST segments, T waves, and... Don't forget the U waves.

- [P waves \(lesson VII\)](#): are they too wide, too tall, look funny (i.e., are they ectopic), etc.?
- QRS complexes: look for [pathologic Q waves \(lesson IX\)](#), [abnormal voltage \(lesson VIII\)](#), etc.
- [ST segments \(lesson X\)](#): look for abnormal ST elevation and/or depression.
- [T waves \(lesson XI\)](#): look for abnormally inverted T waves.
- [U waves \(lesson XII\)](#): look for prominent or inverted U waves.

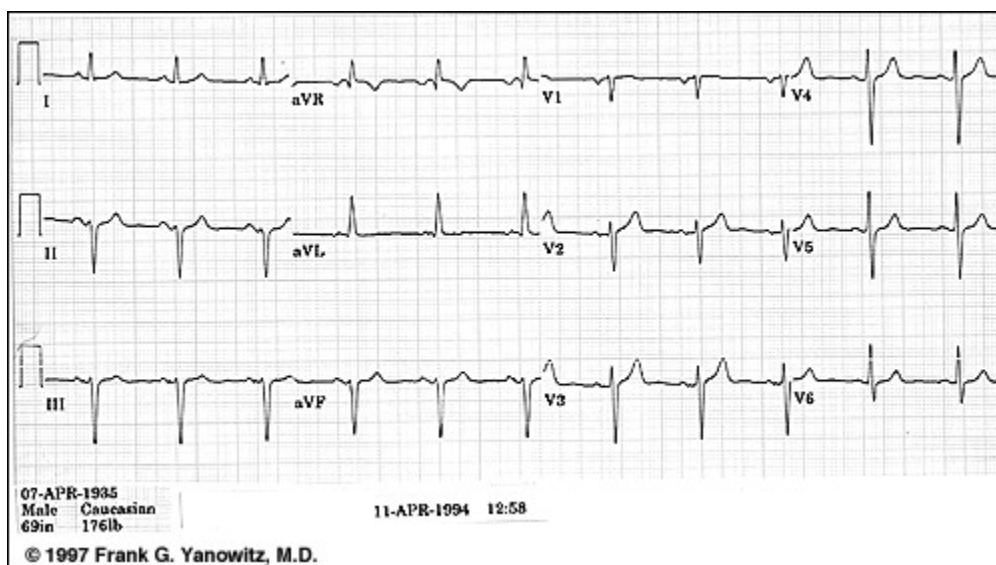
5. ECG Interpretation

➤ This is the conclusion of the above analyses. Interpret the ECG as "Normal", or "Abnormal". Occasionally the term "borderline" is used if unsure about the significance of certain findings. List all abnormalities. Examples of "abnormal" statements are:

- Inferior MI, probably acute
- Old anteroseptal MI
- Left anterior fascicular block (LAFB)
- Left ventricular hypertrophy (LVH)
- Nonspecific ST-T wave abnormalities

● Any rhythm abnormalities

➤ Example:



Left Anterior Fascicular Block (LAFB)-KH

Frank G. Yanowitz, M.D.

HR=72bpm; PR=0.16s; QRS=0.09s; QT=0.36s; QRS axis = -70° (left axis deviation)

Normal sinus rhythm; normal SA and AV conduction; rS in leads II, III, aVF

Interpretation: Abnormal ECG: 1) [Left anterior fascicular block](#)

6. Comparison with previous ecg

➤ If there is a previous ECG in the patient's file, the current ECG should be compared with it to see if any significant changes have occurred. These changes may have important implications for clinical management decisions.

III. Characteristics of the Normal ECG

It is important to remember that there is a wide range of normal variability in the 12 lead ECG. The following "normal" ECG characteristics, therefore, are not absolute. It takes considerable ECG reading experience to discover all the normal variants. Only by following a structured ["Method of ECG Interpretation" \(Lesson II\)](#) and correlating the various ECG findings with the particular patient's clinical status will the ECG become a valuable clinical tool.

Topics for Study:

[Measurements](#)

[Rhythm](#)

[Conduction](#)

[Waveform description](#)

1. Measurements

➤ Heart Rate: 60 - 90 bpm

● [How to calculate the heart rate on ECG paper](#)

➤ PR Interval: 0.12 - 0.20 sec

➤ QRS Duration: 0.06 - 0.10 sec

➤ QT Interval ($QT_c \leq 0.40$ sec)

● **Bazett's Formula:** $QT_c = (QT)/\text{SqRoot RR (in seconds)}$

● **Poor Man's Guide** to upper limits of QT: For HR = 70 bpm, $QT \leq 0.40$ sec; for every 10 bpm increase above 70 subtract 0.02 sec, and for every 10 bpm decrease below 70 add 0.02 sec. For example:

■ $QT \leq 0.38$ @ 80 bpm

■ $QT \leq 0.42$ @ 60 bpm

➤ Frontal Plane QRS Axis: $+90^\circ$ to -30° (in the adult)

2. Rhythm:

Normal sinus rhythm

The P waves in leads I and II must be upright (positive) if the rhythm is coming from the sinus node.

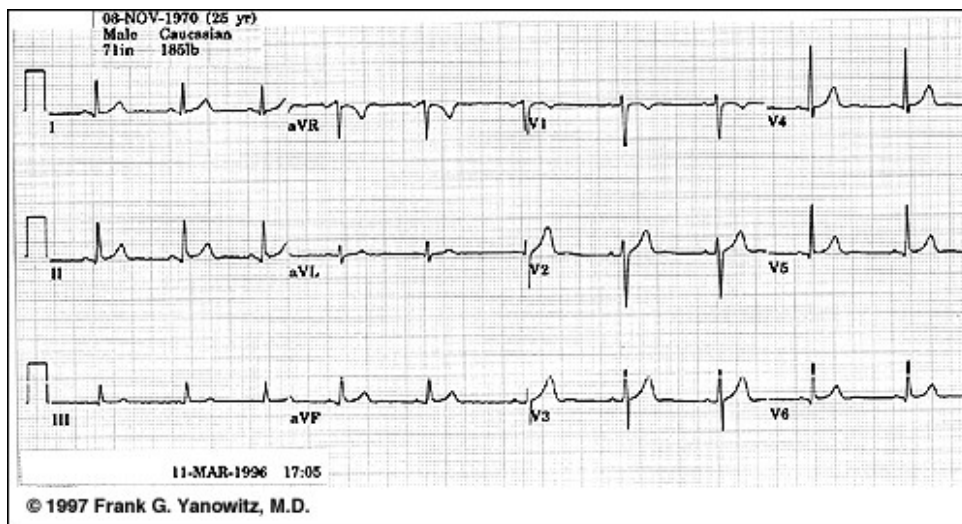
3. Conduction:

Normal Sino-atrial (SA), Atrio-ventricular (AV), and Intraventricular (IV) conduction

Both the PR interval and QRS duration should be within the limits specified above.

4. Waveform Description:

(Normal ECG is shown below - Compare its waveforms to the descriptions below)



➤ P Wave

It is important to remember that the P wave represents the *sequential* activation of the right and left atria, and it is common to see notched or biphasic P waves of right and left atrial activation.

- P duration < 0.12 sec
- P amplitude < 2.5 mm
- Frontal plane P wave axis: 0° to +75°
- May see notched P waves in frontal plane

➤ QRS Complex

The QRS represents the *simultaneous* activation of the right and left ventricles, although most of the QRS waveform is derived from the larger left ventricular musculature.

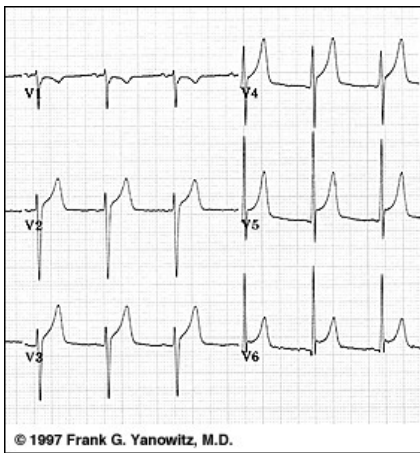
- QRS duration ≤ 0.10 sec
- QRS amplitude is quite variable from lead to lead and from person to person. Two determinates of QRS voltages are:
 - Size of the ventricular chambers (i.e., the larger the chamber, the larger the voltage)
 - Proximity of chest electrodes to ventricular chamber (the closer, the larger the voltage)
- Frontal plane leads:
 - The normal QRS axis range (+90 ° to -30 °); this implies that the QRS be mostly positive (upright) in leads II and I.
 - Normal q-waves reflect normal septal activation (beginning on the LV septum); they are narrow (<0.04s duration) and small (<25% the amplitude of the R wave). They are often seen in leads I and aVL when the QRS axis is to the **left** of +60°, and in leads II, III, aVF when the QRS axis is to the **right** of +60°. Septal q waves should not be confused with the pathologic Q waves of myocardial infarction.
- Precordial leads: (see [Normal ECG](#))
 - Small r-waves begin in V1 or V2 and progress in size to V5. The R-V6 is usually smaller than R-V5.
 - In reverse, the s-waves begin in V6 or V5 and progress in size to V2. S-V1 is usually smaller than S-V2.
 - The usual transition from S>R in the right precordial leads to R>S in the left precordial leads is V3 or V4.
 - Small "septal" q-waves may be seen in leads V5 and V6.

➤ ST Segment and T wave

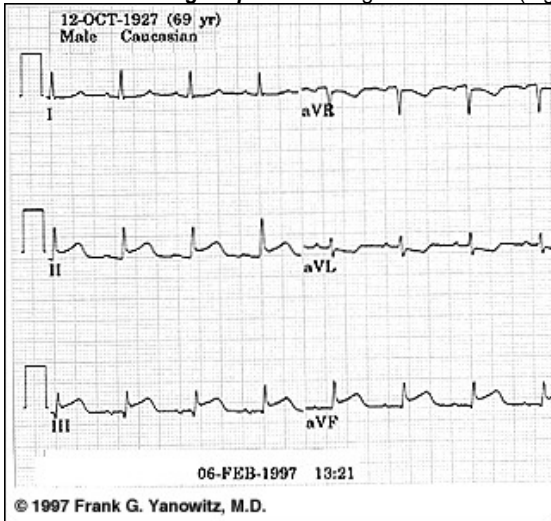
In a sense, the term "ST segment" is a misnomer, because a discrete ST segment distinct from the T wave is usually absent. More often the ST-T wave is a smooth, continuous waveform beginning with the J-point (end of QRS), slowly rising to the peak of the T and followed by a rapid descent to the isoelectric baseline or the onset of the U wave. This gives rise to an asymmetrical T wave. In some normal individuals, particularly women, the T wave is symmetrical and a distinct, horizontal ST segment is present.

The normal T wave is usually in the same direction as the QRS except in the right precordial leads. In the normal ECG the T wave is always upright in leads I, II, V3-6, and always inverted in lead aVR.

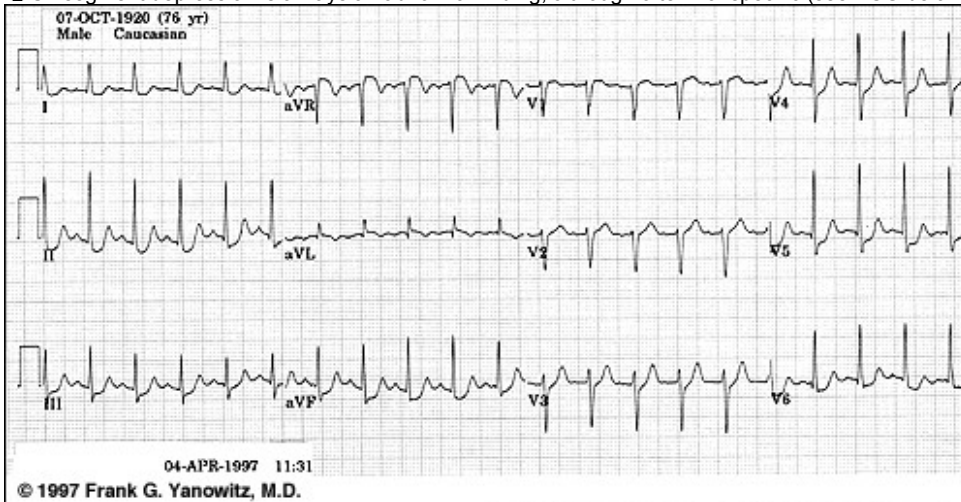
- Normal ST segment elevation: this occurs in leads with large S waves (e.g., V1-3), and the normal configuration is **concave upward**. ST segment elevation with concave upward appearance may also be seen in other leads; this is often called **early repolarization**, although it's a term with little physiologic meaning (see example of "early repolarization" in leads V4-6):



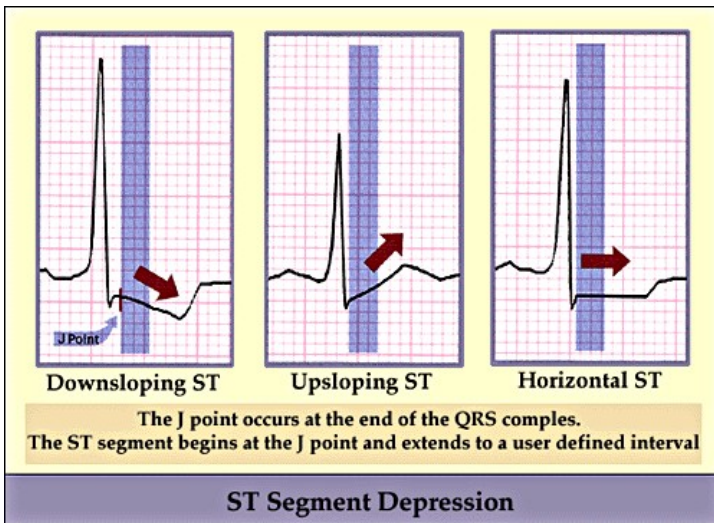
● **Convex or straight upward** ST segment elevation (e.g., leads II, III, aVF) is abnormal and suggests transmural injury or infarction:



● ST segment depression is always an abnormal finding, although often nonspecific (see ECG below):



● ST segment depression is often characterized as "upsloping", "horizontal", or "downsloping".



- The normal U Wave: (the most neglected of the ECG waveforms)
 - U wave amplitude is usually $< 1/3$ T wave amplitude in same lead
 - U wave direction is the same as T wave direction in that lead
 - U waves are more prominent at slow heart rates and usually best seen in the right precordial leads.
 - Origin of the U wave is thought to be related to *afterdepolarizations* which interrupt or follow repolarization.

IV. Abnormalities in the ECG Measurements

Click on the measurement abnormality you would like to study

[Heart Rate](#)
[PR Interval](#)
[QRS Duration](#)
[QT Interval](#)
[QRS Axis](#)

1. Heart Rate

In normal sinus rhythm, a resting heart rate of below 60 bpm is called bradycardia and a rate of above 90 bpm is called tachycardia.

2. PR Interval

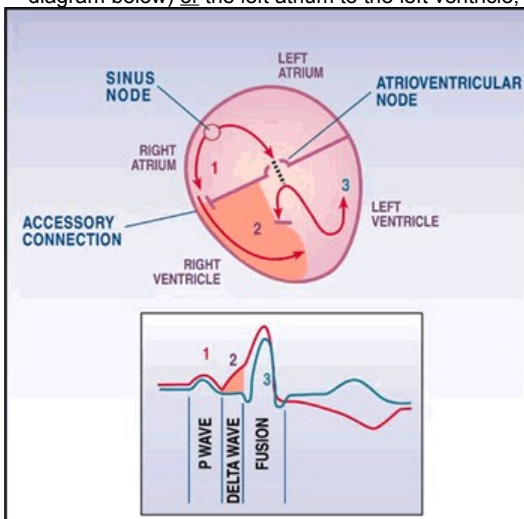
(measured from beginning of P to beginning of QRS in the frontal plane)

➤ Normal: 0.12 - 0.20s

➤ Short PR: $< 0.12s$

● Preexcitation syndromes:

■ WPW (Wolff-Parkinson-White) Syndrome: An accessory pathway (called the "Kent" bundle) connects the right atrium to the right ventricle (see diagram below) or the left atrium to the left ventricle, and this permits early activation of the ventricles (*delta* wave) and a short PR interval.



■ LGL (Lown-Ganong-Levine): An AV nodal bypass track into the His bundle exists, and this permits early activation of the ventricles without a *delta*-wave because the ventricular activation sequence is normal.

● AV Junctional Rhythms with retrograde atrial activation (inverted P waves in II, III, aVF): Retrograde P waves may occur *before* the QRS complex (usually with a short PR interval), *in* the QRS complex (i.e., hidden from view), or *after* the QRS complex (i.e., in the ST segment).

● Ectopic atrial rhythms originating near the AV node (the PR interval is short because atrial activation originates close to the AV node; the P wave morphology is different from the sinus P)

● Normal variant

➤ Prolonged PR: >0.20s

● First degree AV block (PR interval usually constant)

■ Intra-atrial conduction delay (uncommon)

■ Slowed conduction in AV node (most common site)

■ Slowed conduction in His bundle (rare)

■ Slowed conduction in bundle branch (when contralateral bundle is blocked)

● Second degree AV block (PR interval may be normal or prolonged; some P waves do not conduct)

■ Type I (Wenckebach): Increasing PR until nonconducted P wave occurs

■ Type II (Mobitz): Fixed PR intervals plus nonconducted P waves

● AV dissociation: Some PR's may appear prolonged, but the P waves and QRS complexes are dissociated (i.e., not married, but strangers passing in the night).

3. QRS Duration

(duration of QRS complex in frontal plane):

➤ Normal: 0.06 - 0.10s

➤ Prolonged QRS Duration (>0.10s):

● QRS duration 0.10 - 0.12s

■ Incomplete right or left bundle branch block

■ Nonspecific intraventricular conduction delay (IVCD)

■ Some cases of left anterior or posterior fascicular block

● QRS duration $\geq 0.12s$

■ Complete RBBB or LBBB

■ Nonspecific IVCD

■ Ectopic rhythms originating in the ventricles (e.g., ventricular tachycardia, pacemaker rhythm)

4. QT Interval

(measured from beginning of QRS to end of T wave in the frontal plane)

➤ Normal: heart rate dependent (corrected QT = $QT_c = \text{measured QT} \div \text{sq-root RR in seconds}$; upper limit for $QT_c = 0.44 \text{ sec}$)

➤ Long QT Syndrome - "LQTS" (based on upper limits for heart rate; $QT_c \geq 0.47 \text{ sec}$ for males and $\geq 0.48 \text{ sec}$ in females is diagnostic for *hereditary* LQTS in absence of other causes of increased QT)

● This abnormality may have important clinical implications since it usually indicates a state of increased vulnerability to malignant ventricular arrhythmias, syncope, and sudden death. The prototype arrhythmia of the *Long QT Interval Syndromes (LQTS)* is Torsade-de-pointes, a polymorphic ventricular tachycardia characterized by varying QRS morphology and amplitude around the isoelectric baseline. Causes of LQTS include the following:

■ Drugs (many antiarrhythmics, tricyclics, phenothiazines, and others)

■ Electrolyte abnormalities ($\downarrow K^+$, $\downarrow Ca^{++}$, $\downarrow Mg^{++}$)

■ CNS disease (especially subarachnoid hemorrhage, stroke, trauma)

■ Hereditary LQTS (e.g., Romano-Ward Syndrome)

■ Coronary Heart Disease (some post-MI patients)

5. Frontal Plane QRS Axis

➤ Click here for brief tutorial in [Measuring QRS Axis](#)

➤ Normal: -30 degrees to +90 degrees

➤ Abnormalities in the QRS Axis:

● Left Axis Deviation (LAD): $\geq -30^\circ$ (i.e., lead II is mostly 'negative')

- Left Anterior Fascicular Block (LAFB): rS complex in leads II, III, aVF, small q in leads I and/or aVL, and axis -45° to -90°
- Some cases of inferior MI with Qr complex in lead II (making lead II 'negative')
- Inferior MI + LAFB in same patient (QS or qRS complex in lead II)
- Some cases of LVH
- Some cases of LBBB
- Ostium primum ASD and other endocardial cushion defects
- Some cases of WPW syndrome (large negative *delta* wave in lead II)

● Right Axis Deviation (RAD): $\geq +90^\circ$ (i.e., lead I is mostly 'negative')

■ Left Posterior Fascicular Block (LPFB): rS complex in lead I, qR in leads II, III, aVF (*however, must first exclude, on clinical basis, causes of right heart overload; these will also give same ECG picture of LPFB*)

■ Many causes of right heart overload and pulmonary hypertension

■ High lateral wall MI with Qr or QS complex in leads I and aVL

■ Some cases of RBBB

■ Some cases of WPW syndrome

■ Children, teenagers, and some young adults

● Bizarre QRS axis: $+150^\circ$ to -90° (i.e., lead I and lead II are both negative)

■ Consider limb lead error (usually right and left arm reversal)

■ Dextrocardia

■ Some cases of complex congenital heart disease (e.g., transposition)

■ Some cases of ventricular tachycardia

V. ECG Rhythm Abnormalities

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I II III IV V VI VII VIII IX X XI XII

Topics for Study:

1. [Introduction to rhythm analysis](#)

2. [Supraventricular arrhythmias](#)

● [Premature atrial complexes](#)

● [Premature junctional complexes](#)

● [Atrial fibrillation](#)

● [Atrial flutter](#)

● [Ectopic atrial tachycardia and rhythm](#)

● [Multifocal atrial tachycardia](#)

● [Paroxysmal supraventricular tachycardia](#)

● [Junctional rhythms and tachycardias](#)

3. [Ventricular arrhythmias](#)

● [Premature ventricular complexes \(PVCs\)](#)

● [Aberrancy vs. ventricular ectopy](#)

● [Ventricular tachycardia](#)

● [Differential diagnosis of wide QRS tachycardias](#)

● [Accelerated ventricular rhythms](#)

● [Idioventricular rhythm](#)

● [Ventricular parasystole](#)

Lesson V (cont) Introduction to ECG Rhythm Analysis

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[Return to the beginning of Lesson V](#)

Things to Consider When Analyzing Arrhythmias

Arrhythmias may be seen on 12-lead ECGs or on strips of one or more leads. Some arrhythmias are obvious at first glance and don't require intense analysis. Others, however, are more fun! They require detective work, i.e., logical thinking based on a knowledge of cardiac electrophysiology. The analysis should begin with identifying characteristics of **impulse formation** (if known) as well as **impulse conduction**. Here are some things to think about:

[Descriptors of impulse formation](#) (i.e. the pacemaker)

[Descriptors of impulse conduction](#) (i.e., how it moves through the heart)

1. Descriptors of impulse formation

(i.e. the pacemaker or region of impulse formation)

➤ Site of origin (i.e., where is the abnormal rhythm coming from?)

● Sinus Node (e.g., sinus tachycardia)

● Atria (e.g., PAC)

● AV junction (e.g., junctional escape rhythm)

● Ventricles (e.g., PVC)

➤ Rate (i.e., relative to the "expected rate" for that pacemaker location)

● Accelerated - faster than expected (e.g., accelerated junctional rhythm @ 75bpm)

● Slower than expected (e.g., marked sinus bradycardia @ 40bpm)

● Normal (e.g., junctional escape rhythm)

➤ Regularity of ventricular or atrial response

● Regular (e.g., PSVT)

● Regular irregularity (e.g., ventricular bigeminy)

● Irregular irregularity (e.g., atrial fibrillation or MAT)

● Irregular (e.g., multifocal PVCs)

➤ Onset (i.e., how does the arrhythmia begin?)

● Active onset (i.e., begins prematurely as with PAC or PVC)

● Passive onset (e.g., ventricular escape beat or rhythm)

2. Descriptors of impulse conduction

(i.e., how abnormal rhythm conducts through the heart)

➤ Antegrade (forward) vs. retrograde (backward) conduction

➤ Conduction delays or blocks: i.e., 1st, 2nd (type I or II), 3rd degree blocks

➤ Sites of potential conduction delay

- Sino-Atrial (SA) exit block (can only recognize 2nd degree SA block on ECG)
- Intra-atrial delay (usually not recognized)
- AV conduction delays (common)
- IV blocks (e.g., bundle branch or fascicular blocks)

Now let's explore some real rhythm abnormalities..... [\(Return to Lesson V\)](#)

Lesson V (cont) Supraventricular Arrhythmias

[Return to the beginning of Lesson V](#)

1. [Premature atrial complexes](#)
2. [Premature junctional complexes](#)
3. [Atrial fibrillation](#)
4. [Atrial flutter](#)
5. [Ectopic atrial tachycardia and rhythm](#)
6. [Multifocal atrial tachycardia](#)
7. [Paroxysmal supraventricular tachycardia](#)
8. [Junctional rhythms and tachycardias](#)

1. Premature atrial complexes

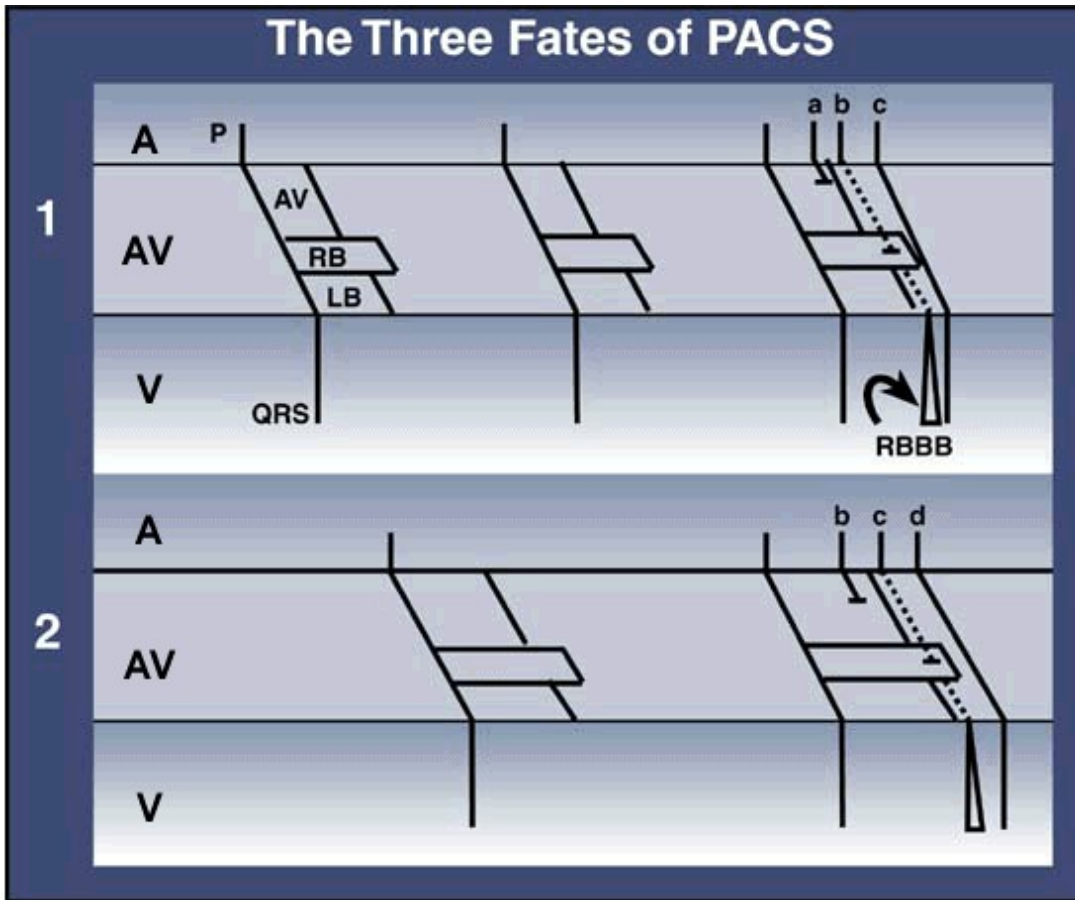
➤ Occur as single or repetitive events and have unifocal or multifocal origins.

➤ The ectopic P wave (called P') is often hidden in the ST-T wave of the preceding beat. (Dr. Marriott, master ECG teacher and author, likes to say: "*Cherchez le P on let T*" which in French means: "Search for the P on the T wave", but it's more sexy in French!)

➤ The P-R interval is normal or prolonged because the AV junction is often partially refractory when the premature impulse enters it.

➤ PAC's can have three different outcomes depending on the degree of prematurity (i.e., coupling interval from previous P wave), and the preceding cycle length. This is illustrated in the "ladder" diagram where normal sinus beats (P) are followed by three possible PACs; in the diagram the refractory periods of the AV node and bundle branches are indicated by the width of the boxes):

The Three Fates of PACS



A "ladder" diagram is an easy way of conceptualizing the conduction of impulses through the heart, and the resulting complexes (i.e., P waves and QRS waves).

● Outcome #1. Nonconducted (blocked); i.e., no QRS complex because the PAC finds AV node still refractory. (see PAC labeled 'a' in the upper diagram 1)

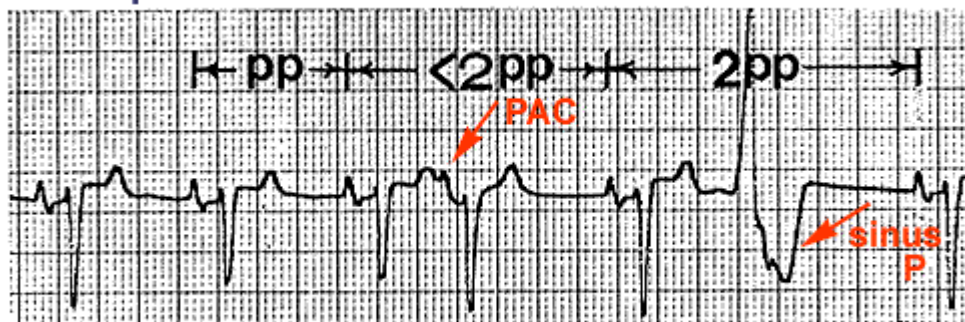
● Outcome #2. Conducted with aberration; i.e., PAC makes it into the ventricles but finds one or more of the conducting fascicles or bundle branches refractory. The resulting QRS is usually wide, and is sometimes called an *Ashman beat* (see PAC 'b' in diagram 1)

● Outcome #3. Normal conduction; i.e., similar to other QRS complexes in the ECG. (See PAC 'c' in the diagram 1)

● In the diagram 2, seen above, the cycle length (i.e., PP interval) has increased (slower heart rate), and this results in increased refractoriness of all the structures in the conduction system (i.e., wider boxes). PAC 'b' now can't get through the AV node and is nonconducted; PAC 'c' is now blocked in the right bundle branch and results in a RBBB QRS complex (aberrant conduction); PAC 'd' is far enough away to conduct normally. *Therefore, the fate of a PAC depends on 1) the coupling interval from the last P wave and 2) the preceding cycle length or heart rate.*

➤ The pause after a PAC is usually *incomplete*; i.e., the PAC usually enters the sinus node and resets its timing, causing the next sinus P to appear earlier than expected. (PVCs, on the other hand, are usually followed by a *complete* pause because the PVC does not usually perturb the sinus node; see ECG below.)

Lead V₁

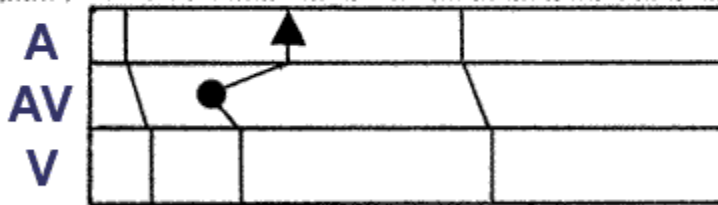


Complete vs Incomplete Pause

2. Premature junctional complexes

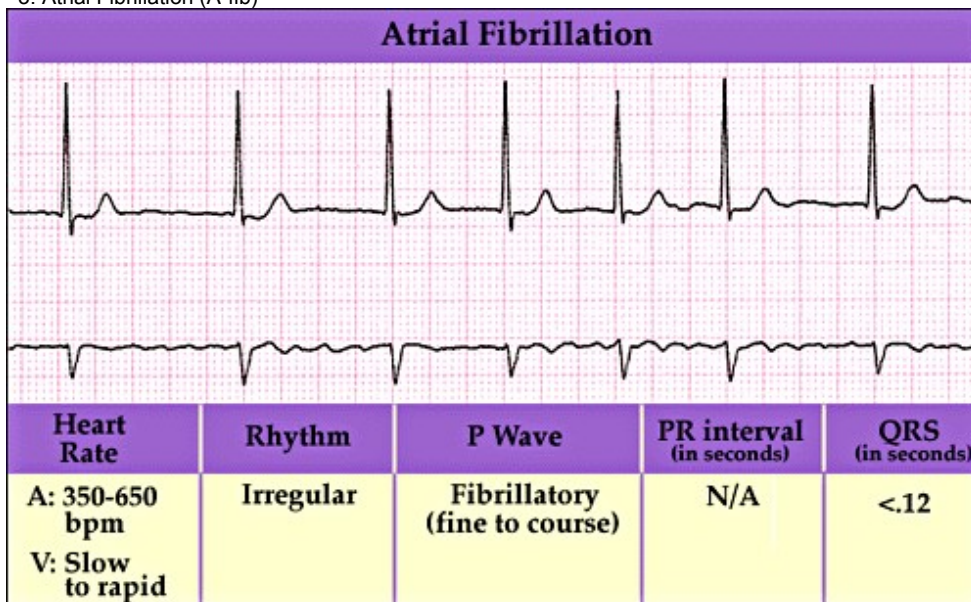
➤ Similar to PAC's in clinical implications, but occur less frequently.

➤ The PJC focus, located in the AV junction, captures the atria (retrograde) and the ventricles (antegrade). The retrograde P wave may appear before, during, or after the QRS complex; if before, the PR interval is usually short (i.e., <0.12 s). The ECG tracing and ladder diagram shown below illustrates two classic PJC's with retrograde P waves following the QRS.



Premature junctional complexes (PJC's)

3. Atrial Fibrillation (A-fib)

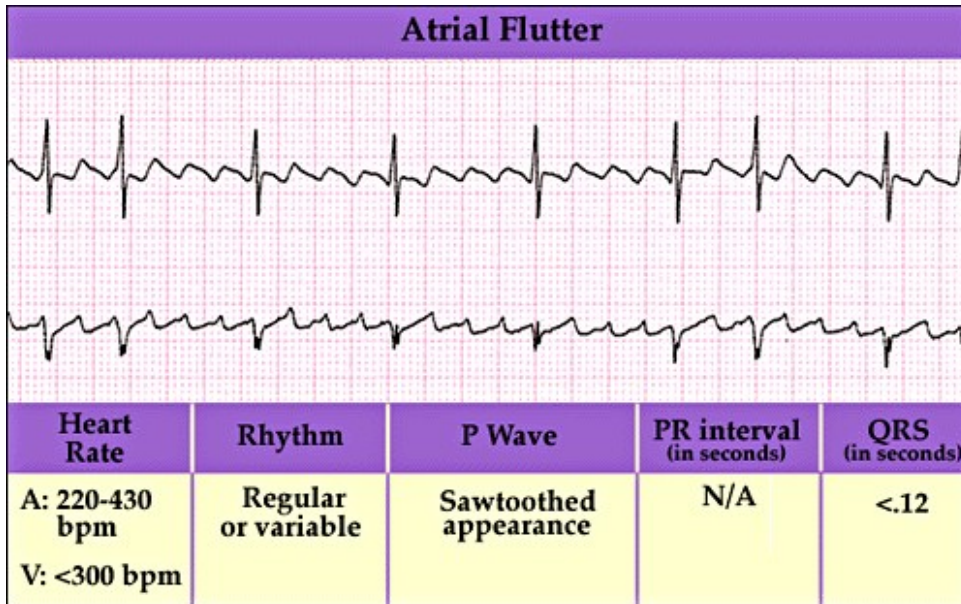


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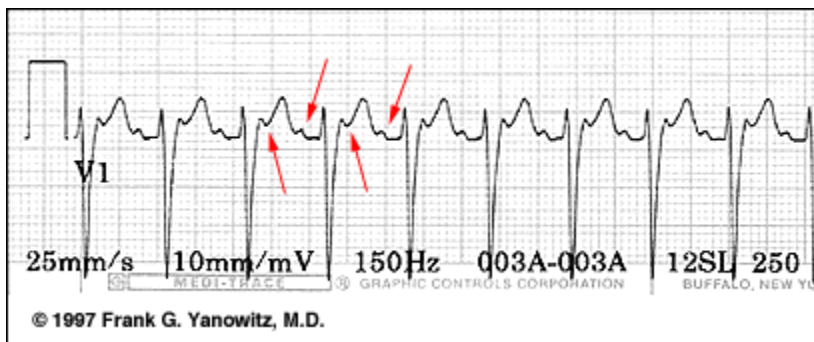
➤ Atrial activity is poorly defined; may see course or fine undulations or no atrial activity at all. If atrial activity is seen, it resembles an *old saw* (when compared to atrial flutter that often resembles a *new saw*).

- Ventricular response is *irregularly irregular* and may be fast (HR >100 bpm, indicates inadequate rate control), moderate (HR = 60-100 bpm), or slow (HR <60 bpm, indicates excessive rate control, AV node disease, or drug toxicity).
- A *regular* ventricular response with A-fib usually indicates complete AV block with an escape or accelerated ectopic pacemaker originating in the AV junction or ventricles (i.e., must consider digoxin toxicity or AV node disease).
- The differential diagnosis includes atrial flutter with an irregular ventricular response *and* multifocal atrial tachycardia (MAT), which is usually irregularly irregular. The differential diagnosis may be hard to make from a single lead rhythm strip; the 12-lead ECG is best for differentiating these three arrhythmias.

4. Atrial Flutter (A-flutter):



- Regular atrial activity with a "clean" *saw-tooth* appearance in leads II, III, aVF, and usually discrete 'P' waves in lead V1. The atrial rate is usually about 300/min, but may be as slow as 150-200/min or as fast as 400-450/min.
- Untreated A-flutter often presents with a 2:1 A-V conduction ratio. This is the most commonly missed supraventricular tachycardia because the flutter waves are often difficult to find when there is 2:1 ratio. Therefore, always think "atrial flutter with 2:1 block" whenever there is a regular supraventricular tachycardia @ ~150 bpm! (You won't miss it if you look for it in a 12-lead ECG)



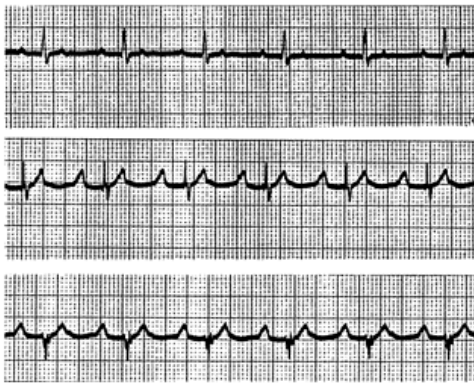
In this ECG rhythm strip, arrows point to atrial flutter waves @ 280bpm with ventricular rate @ 140bpm (atrial flutter with 2:1 block)

- The ventricular response may be 2:1, 3:1 (rare), 4:1, or irregular depending upon the AV conduction properties and AV node slowing drugs on board (e.g., digoxin, beta blockers).

5. Ectopic Atrial Tachycardia and Rhythm

- Ectopic, discrete looking, unifocal P' waves with atrial rate <250/min (not to be confused with slow atrial flutter)
- Ectopic P' waves usually precede QRS complexes with P'R interval < RP' interval (i.e., not to be confused with paroxysmal supraventricular tachycardia with retrograde P waves appearing shortly after the QRS complexes).
- Ventricular response may be 1:1 or with varying degrees of AV block (especially in digitalis toxicity, as shown in this 3-lead ECG with 2:1 block).

Digitalis Toxicity



Atrial tachycardia, 2:1 AV block

1. The P-wave axis is normal
2. The non-conducted P-wave hides in the T-wave
3. The conducted P-wave often has a long PR interval
4. The P-P interval may not be exactly regular

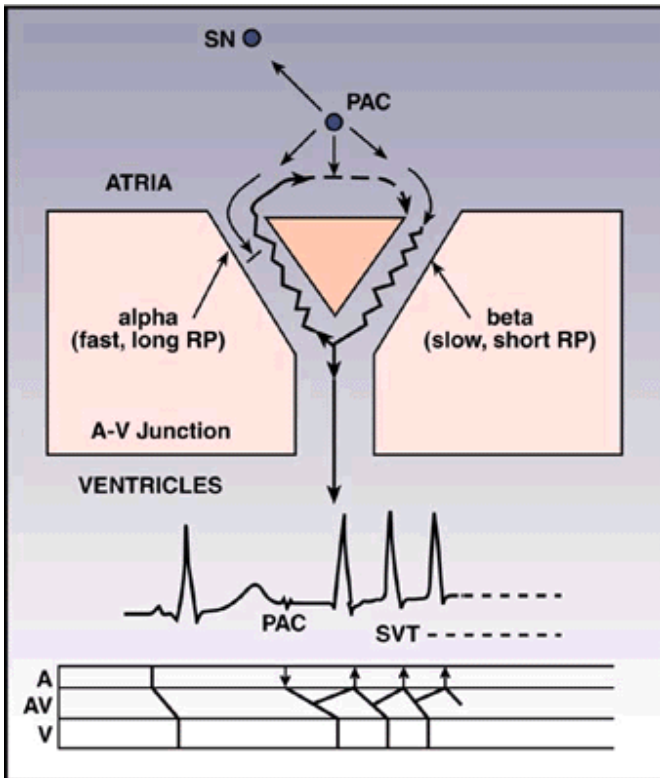
➤ Ectopic atrial rhythm is similar to ectopic atrial tachycardia, but with HR < 100 bpm.

6. Multifocal Atrial Tachycardia (MAT) and rhythm

- Discrete, multifocal P' waves occurring at rates of 100-250/min and with varying P'R intervals (should see at least 3 different P wave morphologies in a given lead).
- Ventricular response is irregularly irregular (i.e., often confused with A-fib).
- May be intermittent, alternating with periods of normal sinus rhythm.
- Seen most often in elderly patients with chronic or acute medical problems such as exacerbation of chronic obstructive pulmonary disease.
- If atrial rate is <100 bpm, call it *multifocal atrial rhythm*

7. Paroxysmal Supraventricular Tachycardia (PSVT)

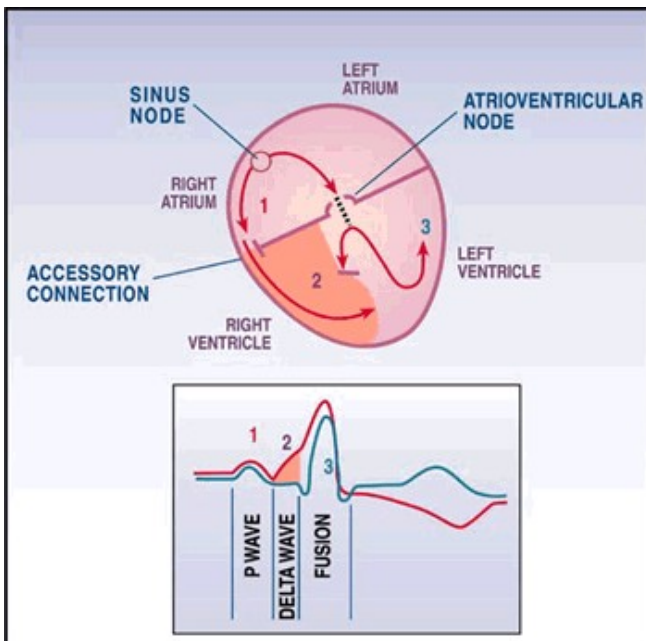
- Basic Considerations: These arrhythmias are *circus movement* or *reciprocating* tachycardias because they utilize the mechanism of *reentry*. The onset is sudden, usually initiated by a premature beat, and the arrhythmia also stops abruptly - which is why they are called *paroxysmal*. They are usually narrow-QRS tachycardias unless there is preexisting bundle branch block or rate-related aberrant ventricular conduction. There are several types of PSVT depending on the location of the reentry circuit.
- AV Nodal Reentrant Tachycardia (AVNRT): This is the most common form of PSVT accounting for approximately 50% of all symptomatic PSVTs. The diagram illustrates the probable mechanism involving dual AV nodal pathways, *alpha* and *beta*, with different electrical properties. In the diagram *alpha* is a fast AV nodal pathway with a long refractory period (RP), and *beta* is the slow pathway with a short RP. During sinus rhythm *alpha* is always used because it conducts faster. An early PAC, however, finds *alpha* still refractory and must use the slower *beta* pathway to reach the ventricles. By the time it traverses *beta*, however, *alpha* has recovered allowing retrograde conduction back to the atria. The retrograde P wave (called an *atrial echo* for obvious reasons) is often simultaneous with the QRS and, therefore, not seen on the ECG, but it can reenter the AV junction because of *beta*'s short RP.



[click here to view](#)

If conditions are right, a circus movement or reciprocating tachycardia results as seen in the above ECG and ladder diagram. Rarely, an "uncommon" form of AVNRT occurs with the retrograde P wave appearing in front of the next QRS (i.e., RP' interval > 1/2 RR interval), implying antegrade conduction down the faster *alpha*, and retrograde conduction up the slower *beta*.

➤ AV Reciprocating Tachycardia (Extranodal bypass pathway): This is the second most common form of PSVT and is seen in patients with WPW syndrome. The WPW ECG, seen in the diagram, shows a short PR, *delta* wave, and somewhat widened QRS.

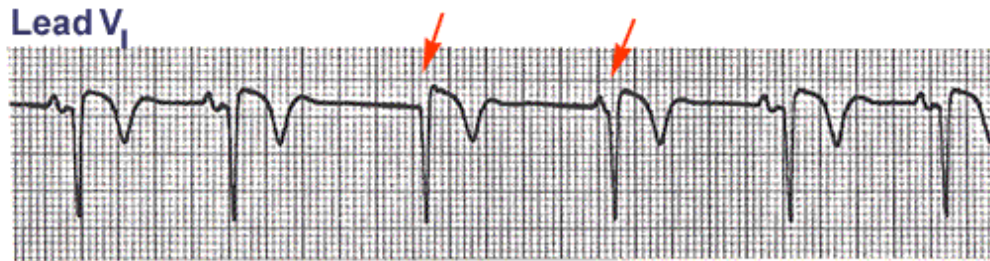


This type of PSVT can also occur in the absence of manifest WPW on a preceding ECG if the accessory pathway only allows conduction in the retrograde direction (i.e., *concealed* WPW). Like AVNRT, a PAC that finds the bypass track temporarily refractory usually initiates the onset of PSVT. The PAC conducts down the normal AV pathway to the ventricles, and reenters the atria retrogradely through the bypass track. In this type of PSVT retrograde P waves appear shortly after the QRS in the ST segment (i.e., RP' < 1/2 RR interval). Rarely the antegrade limb for PSVT uses the bypass track and the retrograde limb uses the AV junction; the PSVT then resembles a wide QRS tachycardia and must be differentiated from ventricular tachycardia.

➤ Sino-Atrial Reentrant Tachycardia: This is a rare form of PSVT where the reentrant circuit is between the sinus node and the right atria. The ECG looks like sinus tachycardia, but the tachycardia is paroxysmal; i.e., it starts and ends abruptly.

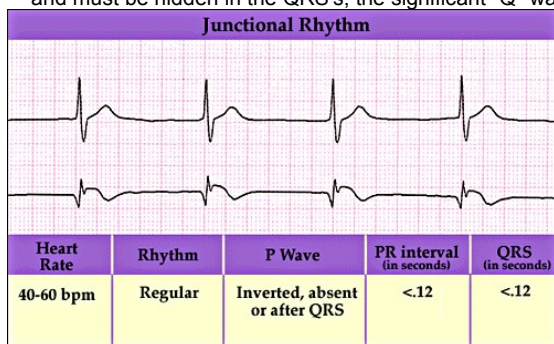
8. Junctional Rhythms and Tachycardias

➤ **Junctional Escape Beats:** These are *passive*, protective beats originating from subsidiary pacemaker cells in the AV junction (usually in the Bundle of His). The pacemaker's basic firing rate is 40-60 bpm; junctional escapes are protective events that occur whenever the primary pacemaker (i.e., sinus node) defaults or the AV node blocks the atrial impulse. The ECG strip shows intermittent sinus slowing with two junctional escapes.

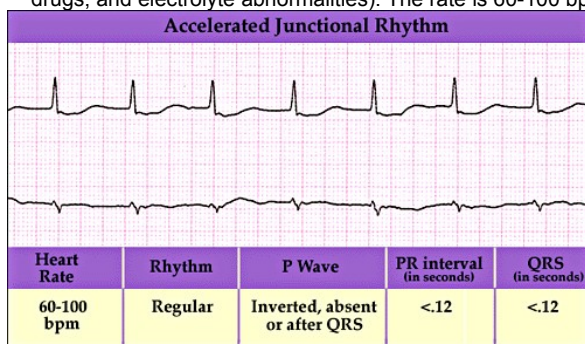


Incomplete AV dissociation due to sinus slowing (default) with junctional escapes (arrows)

➤ **Junctional Escape Rhythm:** This is a sequence of 3 or more junctional escapes occurring by default at a rate of 40-60 bpm. There may be AV dissociation or the atria may be captured retrogradely by the junctional pacemaker. In the ECG example below the retrograde P waves are not seen and must be hidden in the QRS's; the significant "Q" wave with ST elevation in the bottom strip suggests an acute MI.

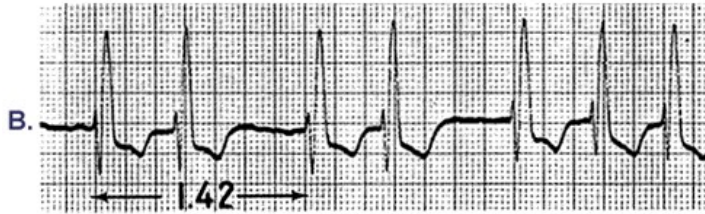
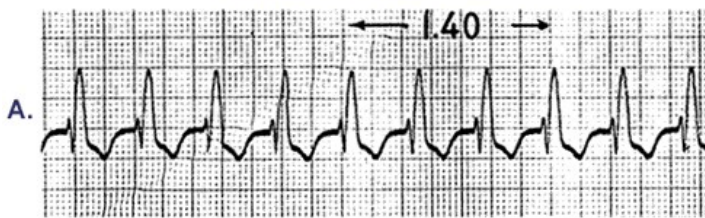


➤ **Accelerated Junctional Rhythm:** This is an *active* junctional pacemaker rhythm caused by events that perturb pacemaker cells (e.g., ischemia, drugs, and electrolyte abnormalities). The rate is 60-100 bpm).



➤ **Nonparoxysmal Junctional Tachycardia:** This usually begins as an accelerated junctional rhythm but the heart rate gradually increases to >100 bpm. There may be AV dissociation, or retrograde atrial capture may occur. Ischemia (usually from right coronary artery occlusion) and digitalis intoxication are the two most common causes. In the example below junctional tachycardia is seen with ('B') and without exit block ('A').

Diagnosis: Digitalis Intoxication



A. Junctional tachycardia

B. Junctional tachycardia with exit block

Lesson V (cont) Ventricular Arrhythmias

[Premature ventricular complexes \(PVCs\)](#)

[Aberrancy vs. ventricular ectopy](#)

[Ventricular tachycardia](#)

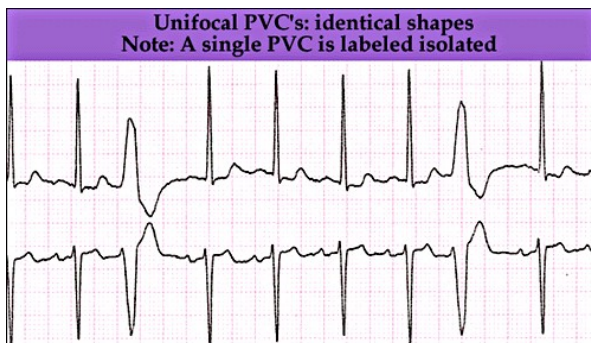
[Differential diagnosis of wide QRS tachycardias](#)

[Accelerated ventricular rhythms](#)

[Idioventricular rhythm](#)

[Ventricular Parasystole](#)

1. Premature Ventricular Complexes (PVCs)



➤ PVCs may be unifocal (see above), multifocal (see below) or multiformed. Multifocal PVCs have different sites of origin, which means their coupling intervals (measured from the previous QRS complexes) are usually different. Multiformed PVCs usually have the same coupling intervals (because they originate in the same ectopic site but their conduction through the ventricles differ. Multiformed PVCs are common in digitalis intoxication.

Multifocal PVC's: more than one shape



➤ PVCs may occur as isolated single events or as couplets, triplets, and salvos (4-6 PVCs in a row), also called brief ventricular tachycardias.

Coupled PVC's: occur in pairs



Triplet PVC's: occur in groups of three



[click here to view](#)

➤ PVCs may occur early in the cycle (R-on-T phenomenon), after the T wave (as seen above), or late in the cycle - often fusing with the next QRS (fusion beat). R-on-T PVCs may be especially dangerous in an acute ischemic situation, because the ventricles may be more vulnerable to ventricular tachycardia or fibrillation. Examples are seen below.

R on T: occur on the peak of the T wave of the preceding beat



Lead V₁

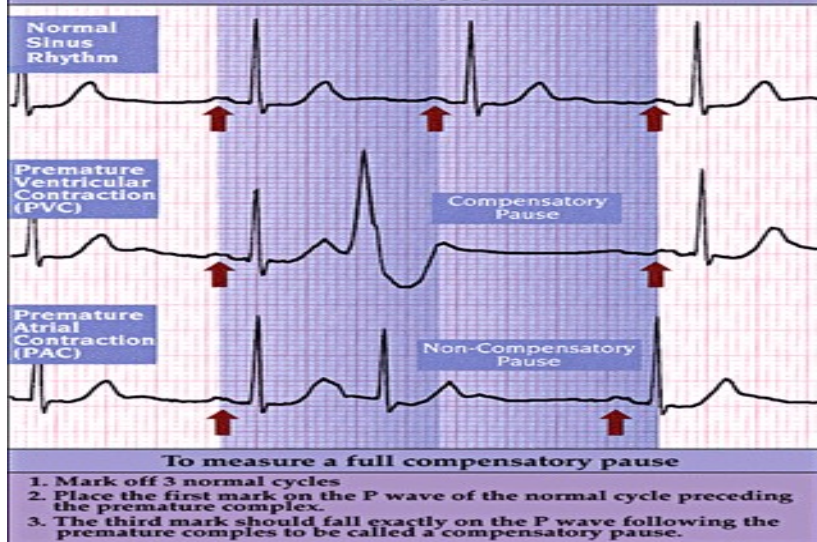


A profusion of fusions! (F)

In the above example, "late" (end-diastolic) PVCs are illustrated with varying degrees of fusion. For fusion to occur the sinus P wave must have made it to the ventricles to start the activation sequence, but before ventricular activation is completed the "late" PVC occurs. The resultant QRS looks a bit like the normal QRS, and a bit like the PVC; i.e., a fusion QRS.

- The events following a PVC are of interest. Usually a PVC is followed by a complete compensatory pause because the sinus node timing is not interrupted; one sinus P wave isn't able to reach the ventricles because they are still refractory from the PVC; the following sinus impulse occurs on time based on the sinus rate. In contrast, PACs are usually followed by an incomplete pause because the PAC usually enters the sinus node and resets its timing; this enables the following sinus P wave to appear earlier than expected. These concepts are illustrated below.

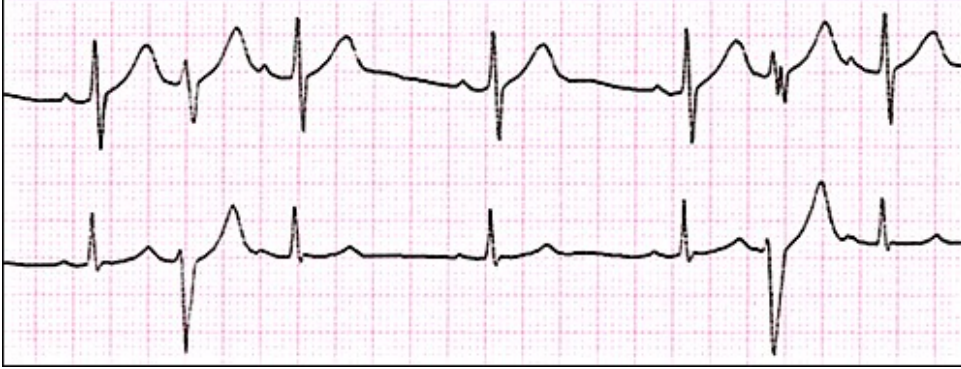
Compensatory vs Noncompensatory Pauses



[click here to view](#)

- Not all PVCs are followed by a pause. If a PVC occurs early enough (especially if the heart rate is slow), it may appear sandwiched in between two normal beats. This is called an interpolated PVC. The sinus impulse following the PVC may be conducted with a longer PR interval because of retrograde concealed conduction by the PVC into the AV junction slowing subsequent conduction of the sinus impulse.

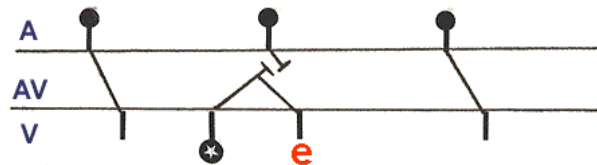
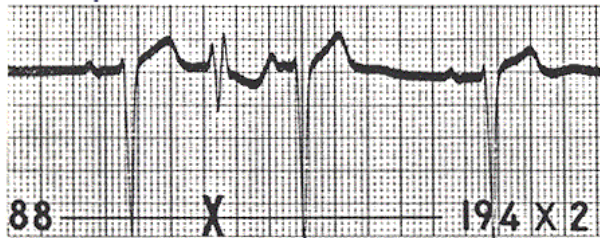
Interpolated PVC's: occur between sinus beats without a compensatory pause



➤ Finally a PVC may retrogradely capture the atrium, reset the sinus node, and be followed by an incomplete pause. Often the retrograde P wave can be seen on the ECG, hiding in the ST-T wave of the PVC.

➤ The most unusual post-PVC event is when retrograde activation of the AV junction re-enters the ventricles as a ventricular echo. This is illustrated below. The "ladder" diagram below the ECG helps us understand the mechanism. The P wave following the PVC is the sinus P wave, but the PR interval is too short for it to have caused the next QRS. (Remember, the PR interval following an interpolated PVC is usually longer than normal, not shorter!).

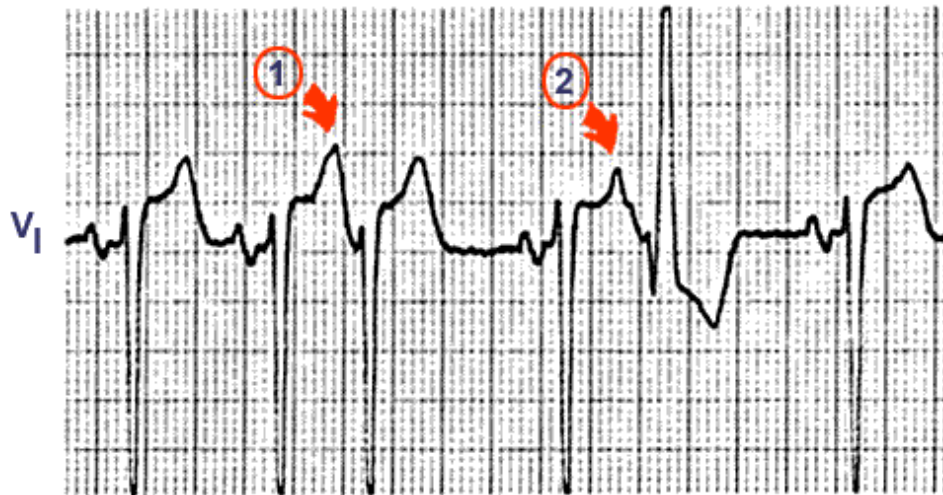
Lead V₁



Interpolated PVC with ventricular echo (e)

➤ PVCs usually stick out like "sore thumbs", because they are bizarre in appearance compared to the normal complexes. However, not all premature sore thumbs are PVCs. In the example below 2 PACs are seen, #1 with a normal QRS, and #2 with RBBB aberrancy - which looks like a sore thumb. **The challenge, therefore, is to recognize sore thumbs for what they are, and that's the next topic for discussion!**

Not all sore thumbs are PVC's



PAC (1) with normal IV conduction

PAC (2) with RBBB aberration (note: longer preceding cycle prior to aberrancy)

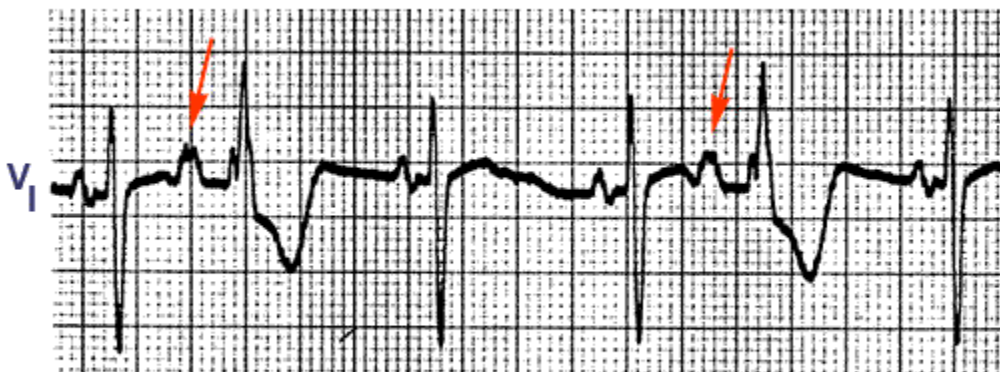
2. Aberrancy vs. Ventricular Ectopy

A most important question

➤ **Aberrant Ventricular Conduction:** defined as the intermittent abnormal intraventricular conduction of a supraventricular impulse. The phenomenon comes about because of unequal refractoriness of the bundle branches and critical prematurity of a supraventricular impulse (see [diagram](#) of "Three Fates of PACs"). With such critical prematurity, the supraventricular impulse encounters one bundle branch (or fascicle) which is responsive, and the other which is refractory, and is consequently conducted with a bundle branch block or fascicular block pattern.

➤ ECG clues to the differential diagnosis of wide QRS premature beats:

- Preceding ectopic P wave (i.e., the P' of the PAC) usually hidden in the ST-T wave of the previous beat favors aberrant ventricular conduction. In the ECG below note the arrow pointing at a premature P wave in the ST-T segment. The QRS has a RBBB morphology.



PAC's (arrow) with atypical RBBB aberration

- Analyze the [compensatory pause](#): A complete pause favors ventricular ectopy (i.e., no resetting of the sinus pacemaker; next sinus impulse comes on time). An incomplete pause favors aberration (i.e., because supraventricular prematures are more likely to reset the sinus node's timing). Be aware of exceptions to this simple rule because PVCs can activate the atria retrogradely and reset the sinus node (incomplete pause), and PACs can fail to reset the sinus node (complete pause).

- Long-Short Rule (Ashman Phenomenon): The earlier in the cycle a PAC occurs and the longer the preceding cycle, the more likely the PAC will be conducted with aberration (see [diagram](#) "The Three Fates of PACs"). This is because the refractory period of the ventricular conduction system is proportional to cycle length or heart rate; the longer the cycle length or slower the heart rate, the longer the recovery time of the conduction system. In most individuals the right bundle normally recovers more slowly than the left bundle, and a critically timed PAC is therefore more likely to conduct with RBBB than with LBBB. In diseased hearts, however, LBBB aberrancy is also seen. Dr. Richard Ashman and colleagues first described

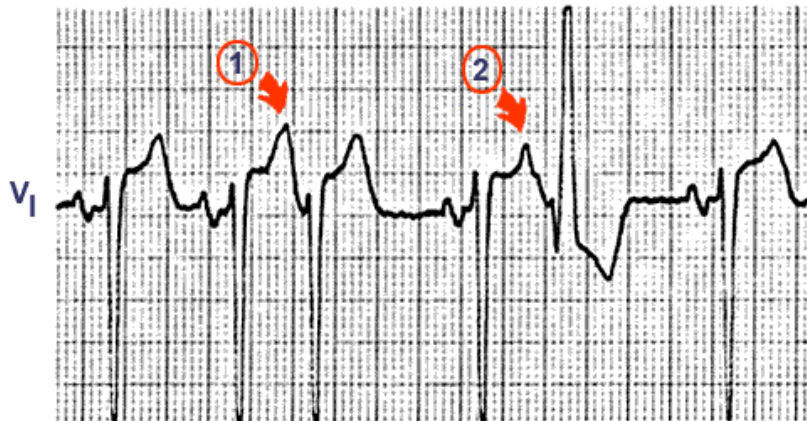
this in 1947 in patients with atrial fibrillation. He noted that the QRS complexes ending a short RR interval were often of a RBBB pattern if the preceding RR interval was long. (That's all it takes to get your name attached to a phenomenon; you must publish!).

● Analyze the **QRS morphology** of the funny-looking beat. This is one of the most rewarding of the clinical clues, especially if lead V1 (or the MCL1 monitored lead in intensive care units) is used. Since aberrancy is almost always in the form of a bundle branch block morphology, V1 is the best lead for differentiating RBBB from LBBB; RBBB creates a positive deflection, and LBBB, a negative deflection. Therefore, the first order of business is to identify the direction of QRS forces in V1.

If the QRS in V1 is mostly positive the following possibilities exist:

■ rsR' or rSR' QRS morphologies suggests RBBB aberrancy >90% of the time!

Not all sore thumbs are PVC's



PAC (1) with normal IV conduction

PAC (2) with RBBB aberration (note: longer preceding cycle prior to aberrancy)

Note the rsR' morphology of PAC #2!

■ monophasic R waves or R waves with a notch or slur on the downstroke of the R waves suggests ventricular ectopy > 90% of the time (see below)!

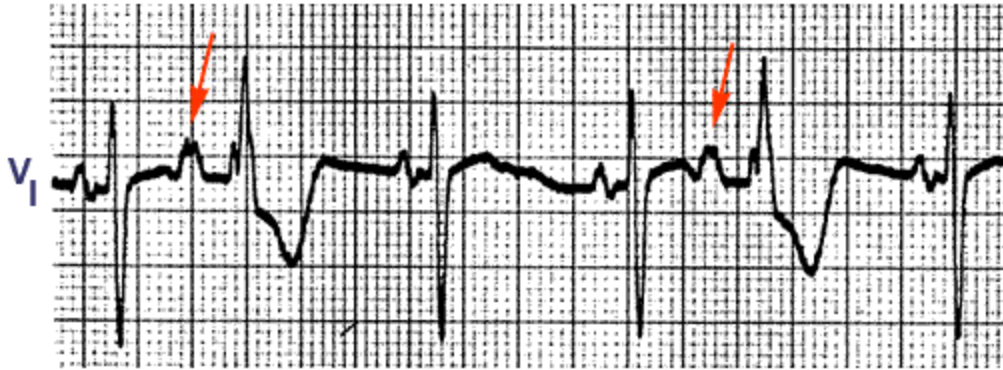
Lead V₁



Atrial fibrillation and PVC's with classic LV morphology

Note position of notch (arrow)

■ monophasic R wave with a notch or slur on the upstroke of R wave: 50-50 possibility or either!



PAC's (arrow) with atypical RBBB aberration

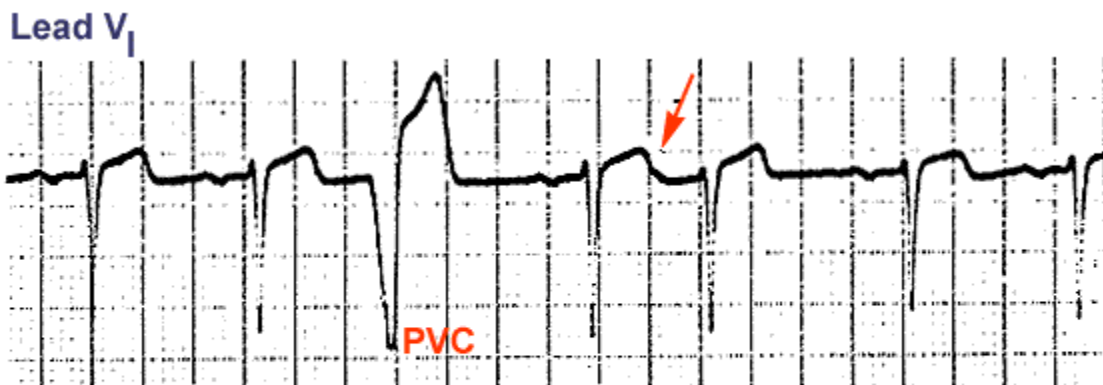
In the above ECG the premature wide QRS is an aberrantly conducted PAC because of the easily seen preceding P wave. The QRS morphology could be either!

■ qR morphology suggests ventricular ectopy unless a previous anteroseptal MI or unless the patient's normal V1 QRS complex has a QS morphology (i.e., no initial r-wave)!

If the QRS in V1 is mostly negative the following possibilities exist:

■ Rapid downstroke of the S wave with or without a preceding "thin" r wave suggests LBBB aberrancy almost always!

■ Fat" r wave (0.04s) or notch/slur on downstroke of S wave or >0.06s delay from QRS onset to nadir of S wave almost always suggests ventricular ectopy!



PAC (arrow) - hidden in T-wave

PVC (RV origin) - sticks out like sore thumb

In the above ECG the wide premature QRS is a PVC because of the >0.06s delay from onset of the QRS to the nadir of the S wave (approximately 0.08s).

Another QRS morphology clue from Lead V6:

■ If the wide QRS morphology is predominately negative in direction in lead V6, then it's most likely ventricular ectopy (assuming V6 is accurately placed in mid axillary line)!

● The timing of the premature wide QRS complex is also important because aberrantly conducted QRS complexes only occur early in the cardiac cycle during the refractory period of one of the conduction branches. Therefore, late premature wide QRS complexes (after the T wave, for example) are most often ventricular ectopic in origin.

3. Ventricular Tachycardia

➤ Descriptors to consider when considering ventricular tachycardia:

- Sustained (lasting >30 sec) vs. nonsustained
- Monomorphic (uniform morphology) vs. polymorphic vs. Torsade-de-pointes
- **Torsade-de-pointes:** a polymorphic ventricular tachycardia associated with the long-QT syndromes characterized by phasic variations in the polarity of the QRS complexes around the baseline. Ventricular rate is often >200bpm and ventricular fibrillation is a consequence.
- Presence of AV dissociation (independent atrial activity) vs. retrograde atrial capture
- Presence of fusion QRS complexes (Dressler beats) which occur when supraventricular beats (usually sinus) get into the ventricles during the ectopic activation sequence.
- **Differential Diagnosis:** just as for single premature funny-looking beats, **not all wide QRS tachycardias are ventricular in origin (i.e., they may be supraventricular tachycardias with bundle branch block or WPW preexcitation)!**

4. Differential Diagnosis of Wide QRS Tachycardias

- Although this is an ECG tutorial, let's not forget some simple bedside clues to ventricular tachycardia:
- Advanced heart disease (e.g., coronary heart disease) statistically favors ventricular tachycardia
- Cannon 'a' waves in the jugular venous pulse suggests ventricular tachycardia with AV dissociation. Under these circumstances atrial contractions may occur when the tricuspid valve is still closed which leads to the giant retrograde pulsations seen in the JV pulse. With AV dissociation these giant a-waves occur irregularly.
- Variable intensity of the S1 heart sound at the apex (mitral closure); again this is seen when there is AV dissociation resulting in varying position of the mitral valve leaflets depending on the timing of atrial and ventricular systole.
- If the patient is hemodynamically unstable, **think ventricular tachycardia** and act accordingly!

➤ ECG Clues:

- **Regularity of the rhythm:** If the wide QRS tachycardia is sustained and monomorphic, then the rhythm is usually regular (i.e., RR intervals equal); an irregularly-irregular rhythm suggests atrial fibrillation with aberration or with WPW preexcitation.

● **A-V Dissociation** strongly suggests ventricular tachycardia! Unfortunately AV dissociation only occurs in approximately 50% of ventricular tachycardias (the other 50% have retrograde atrial capture or "V-A association"). Of the patients with AV dissociation, it is only easily recognized if the rate of tachycardia is <150 bpm. Faster heart rates make it difficult to visualize dissociated P waves.

- **Fusion beats or captures** often occur when there is AV dissociation and this also strongly suggests a ventricular origin for the wide QRS tachycardia.

● **QRS morphology in lead V1 or V6** as described above for single premature funny looking beats is often the best clue to the origin, so go back and check out the clues! Also consider a few other morphology clues:

■ Bizarre frontal-plane QRS axis (i.e. from +150 degrees to -90 degrees or NW quadrant) suggests ventricular tachycardia

■ QRS morphology similar to previously seen PVCs suggests ventricular tachycardia

■ If all the QRS complexes from V1 to V6 are in the same direction (positive or negative), ventricular tachycardia is likely

■ Especially wide QRS complexes (>0.16s) suggests ventricular tachycardia

■ Also consider the following **Four-step Algorithm** reported by *Brugada et al, Circulation 1991;83:1649*:

Step 1: Absence of RS complex in all leads V1-V6?

Yes: Dx is ventricular tachycardia!

Step 2: No: Is interval from beginning of R wave to nadir of S wave >0.1s in any RS lead?

Yes: Dx is ventricular tachycardia!

Step 3: No: Are AV dissociation, fusions, or captures seen?

Yes: Dx is ventricular tachycardia!

Step 4: No: Are there morphology criteria for VT present both in leads V1 and V6?

Yes: Dx is ventricular tachycardia!

NO: Diagnosis is supraventricular tachycardia with aberration!

5. Accelerated Ventricular Rhythms

(see ECG below)

➤ An "active" ventricular rhythm due to enhanced automaticity of a ventricular pacemaker (reperfusion after thrombolytic therapy is a common causal factor).

➤ Ventricular rate 60-100 bpm (anything faster would be ventricular tachycardia)

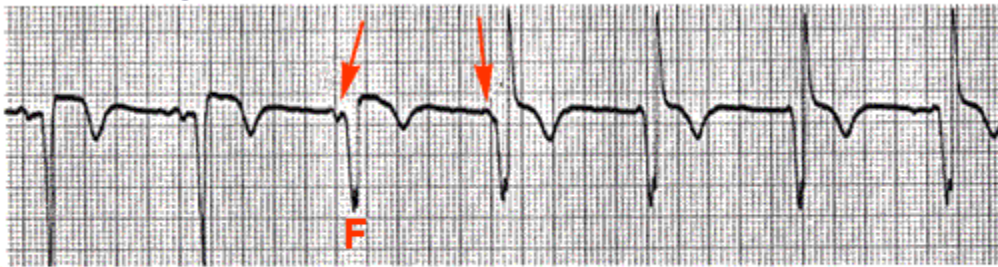
➤ Sometimes called **isochronic ventricular rhythm** because the ventricular rate is close to underlying sinus rate

➤ May begin and end with fusion beats (ventricular activation partly due to the normal sinus activation of the ventricles and partly from the ectopic

focus).

- Usually benign, short lasting, and not requiring of therapy.

Lead MCL_I



Isochronic Ventricular rhythm
F = fusion beat
Arrows point at dissociated P waves

6. Idioventricular Rhythm

- A "passive" escape rhythm that occurs by default whenever higher-level pacemakers in AV junction or sinus node fail to control ventricular activation.
- Escape rate is usually 30-50 bpm (i.e., slower than a junctional escape rhythm).
- Seen most often in complete AV block with AV dissociation or in other bradycardic conditions.

7. Ventricular Parasystole

- Non-fixed coupled PVCs where the inter-ectopic intervals (i.e., timing between PVCs) are some multiple (i.e., 1x, 2x, 3x, . . . etc.) of the basic rate of the parasystolic focus
- PVCs have uniform morphology unless fusion beats occur
- Usually entrance block is present around the ectopic focus, which means that the primary rhythm (e.g., sinus rhythm) is unable to enter the ectopic site and reset its timing.
- May also see exit block; i.e., the output from the ectopic site may occasionally be blocked (i.e., no PVC when one is expected).
- Fusion beats are common when ectopic site fires while ventricles are already being activated from primary pacemaker

Lead V_I



Parasystolic PVC's with fusions (arrows)

- Parasystolic rhythms may also be seen in the atria and AV junction

VI. ECG Conduction Abnormalities

Topics for Study

[Introduction](#)

[Sino-Atrial Exit Block](#)

[Atrio-Ventricular \(AV\) Block](#)

● [1st Degree AV Block](#)

● [Type I \(Wenckebach\) 2nd Degree AV Block](#)

● [Type II \(Mobitz\) 2nd Degree AV Block](#)

● [Complete \(3rd Degree\) AV Block](#)

● [AV Dissociation](#)

[Intraventricular Blocks](#)

● [Right Bundle Branch Block](#)

● [Left Bundle Branch Block](#)

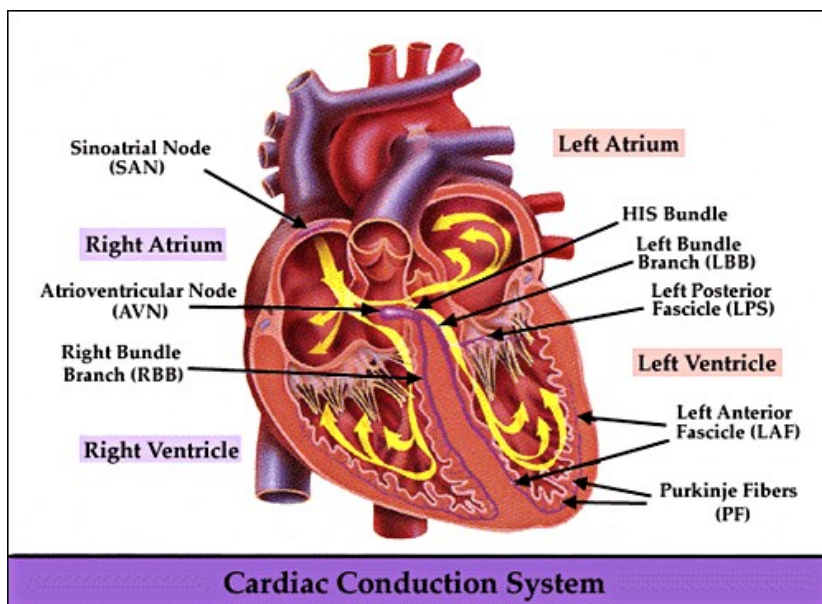
● [Left Anterior Fascicular Block](#)

● [Left Posterior Fascicular Block](#)

● [Bifascicular Blocks](#)

● [Nonspecific Intraventricular Block](#)

● [Wolff-Parkinson-White Preexcitation](#)



[click here to view](#)

1. Introduction:

This section considers all the important disorders of impulse conduction that may occur within the cardiac conduction system illustrated in the above diagram. Heart block can occur anywhere in the specialized conduction system beginning with the sino-atrial connections, the AV junction, the bundle branches and their fascicles, and ending in the distal ventricular Purkinje fibers. Disorders of conduction may manifest as slowed conduction (**1st degree**), intermittent conduction failure (**2nd degree**), or complete conduction failure (**3rd degree**). In addition, 2nd degree heart block occurs in two varieties: Type I (**Wenckebach**) and Type II (**Mobitz**). In Type I block there is **decremental conduction** which means that conduction velocity progressively slows down until failure of conduction occurs. Type II block is all or none. The term **exit block** is used to identify conduction delay or failure immediately distal to a pacemaker site. Sino-atrial (SA) block is an exit block. This section considers conduction disorders in the anatomical sequence that defines the cardiac conduction system; **so lets begin . . .**

2. Sino-Atrial Exit Block (SA Block):

➤ 2nd Degree SA Block: this is the only degree of SA block that can be recognized on the surface ECG (i.e., intermittent conduction failure between the sinus node and the right atrium). There are two types, although because of sinus arrhythmia they may be hard to differentiate. Furthermore, the differentiation is electrocardiographically interesting but not clinically important.

● Type I (SA Wenckebach): the following 3 rules represent the classic rules of Wenckebach, which were originally described for **Type I AV block**. The rules are the result of decremental conduction where the **increment** in conduction delay for each subsequent impulse gets smaller until conduction failure finally occurs. This declining increment results in the following findings:

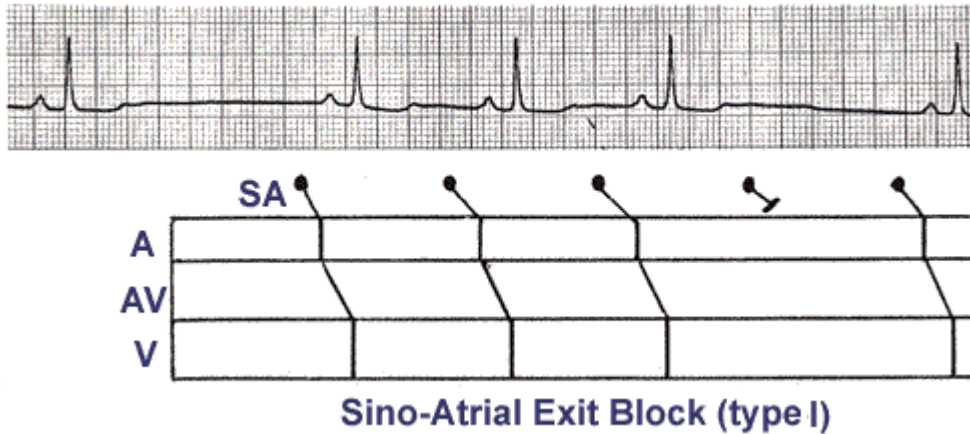
■ PP intervals gradually shorten until a pause occurs (i.e., the blocked sinus impulse fails to reach the atria)

■ The pause duration is **less than** the two preceding PP intervals

■ The PP interval following the pause is **greater than** the PP interval just before the pause

Differential Diagnosis: sinus arrhythmia without SA block. The following rhythm strip illustrates SA Wenckebach with a ladder diagram to show the progressive conduction delay between SA node and the atria. Note the similarity of this rhythm to marked sinus arrhythmia. (Remember, we cannot see SA events on the ECG, only the atrial response or P waves.)

Lead II

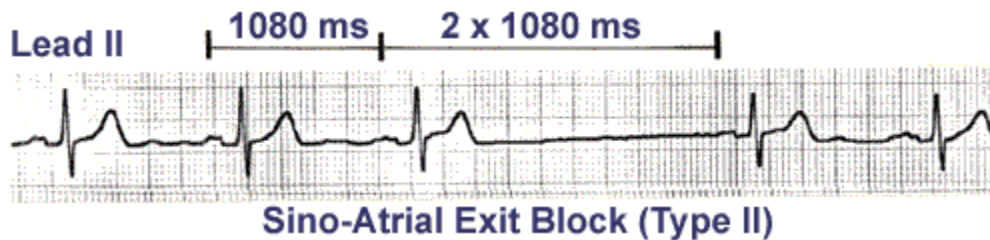


[click here to view](#)

● Type II SA Block:

■ PP intervals fairly constant (unless sinus arrhythmia present) until conduction failure occurs.

■ The pause is approximately **twice** the basic PP interval



3. Atrio-Ventricular (AV) Block

➤ Possible sites of AV block:

● AV node (most common)

● His bundle (uncommon)

● Bundle branch and fascicular divisions (in presence of already existing complete bundle branch block)

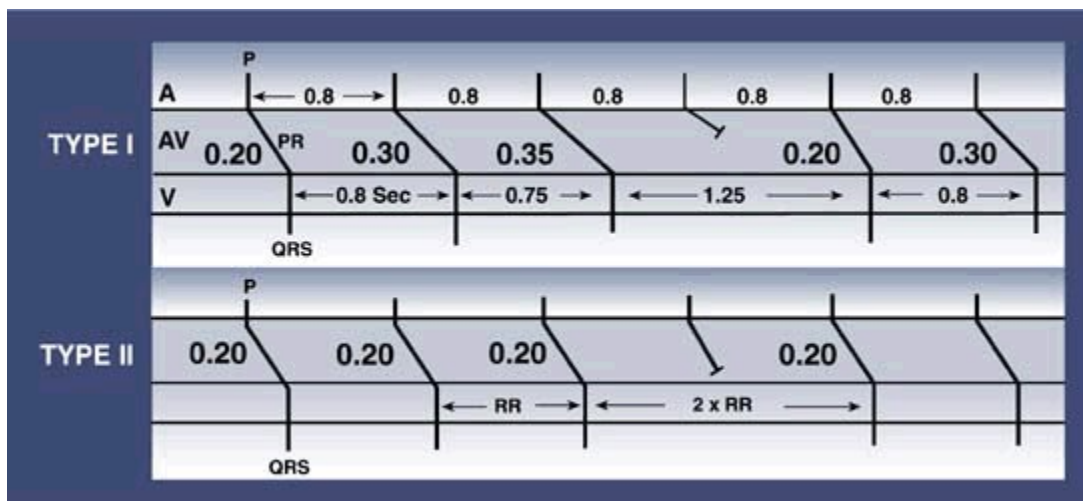
➤ **1st Degree AV Block:** PR interval > 0.20 sec; all P waves conduct to the ventricles.



1st degree AV block (PR = 280 ms)

[click here to view](#)

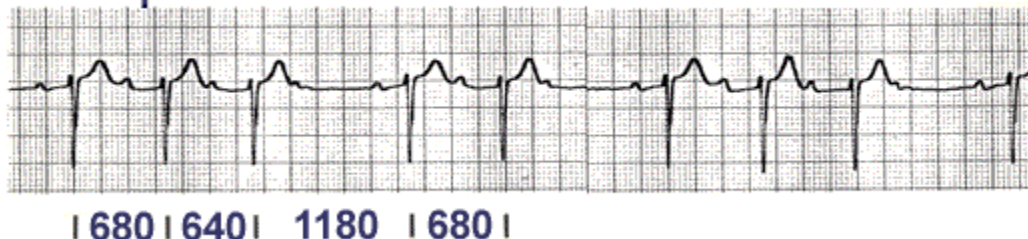
➤ **2nd Degree AV Block:** The diagram below illustrates the difference between Type I (or Wenckebach) and Type II AV block.



In "classic" Type I (**Wenckebach**) AV block the PR interval gets longer (**by shorter increments**) until a nonconducted P wave occurs. The RR interval of the pause is **less than** the two preceding RR intervals, and the RR interval after the pause is **greater than** the RR interval before the pause. These are the **classic** rules of **Wenckebach** (atypical forms can occur). In Type II (**Mobitz**) AV block the PR intervals are constant until a nonconducted P wave occurs. There must be **two consecutive constant** PR intervals to diagnose Type II AV block (i.e., if there is 2:1 AV block we can't be sure if its type I or II). The RR interval of the pause is **equal to** the two preceding RR intervals.

● Type I (Wenckebach) AV block (note the RR intervals in ms duration):

Lead V₁ "Classic Wenckebach"



[click here to view](#)

Type I AV block is **almost always** located in the AV node, which means that the QRS duration is usually narrow, unless there is preexisting bundle branch disease.

● Type II (Mobitz) AV block (note there are two consecutive constant PR intervals before the blocked P wave):

Lead V₁

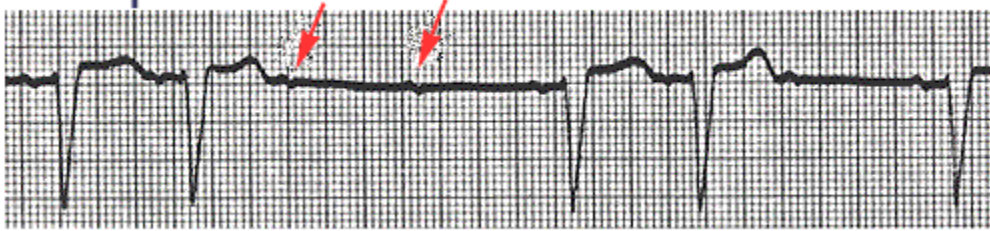


2nd degree AV block (type II) with LBBB

[click here to view](#)

Type II AV block is **almost always** located in the bundle branches, which means that the QRS duration is wide indicating complete block of one bundle; the nonconducted P wave is blocked in the other bundle. In Type II block several consecutive P waves may be blocked as illustrated below:

Lead V₁



➤ Complete (3rd Degree) AV Block

- Usually see **complete AV dissociation** because the atria and ventricles are each controlled by separate pacemakers.
- Narrow QRS rhythm suggests a junctional escape focus for the ventricles with block above the pacemaker focus, usually in the AV node.
- Wide QRS rhythm suggests a ventricular escape focus (i.e., **idioventricular rhythm**). This is seen in ECG 'A' below; ECG 'B' shows the treatment for 3rd degree AV block; i.e., a **ventricular pacemaker**. The location of the block may be in the AV junction or bilaterally in the bundle branches.

Lead V₁

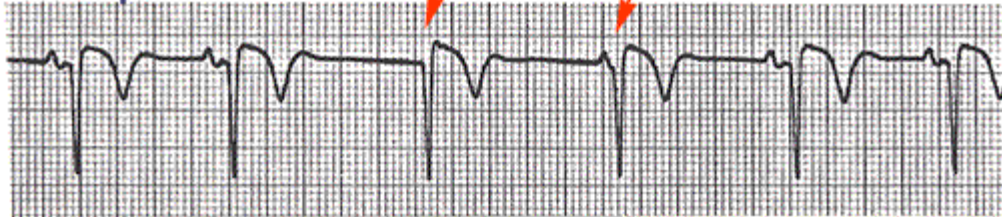


➤ AV Dissociation (independent rhythms in atria and ventricles):

- Not synonymous with 3rd degree AV block, although AV block is one of the causes.
- May be **complete** or **incomplete**. In **complete** AV dissociation the atria and ventricles are always independent of each other. In **incomplete** AV dissociation there is either intermittent atrial *capture* from the ventricular focus or ventricular *capture* from the atrial focus.
- There are **three categories** of AV dissociation (categories 1 & 2 are always **incomplete** AV dissociation):

1. Slowing of the primary pacemaker (i.e., SA node); subsidiary **escape** pacemaker takes over by **default**:

Lead V₁

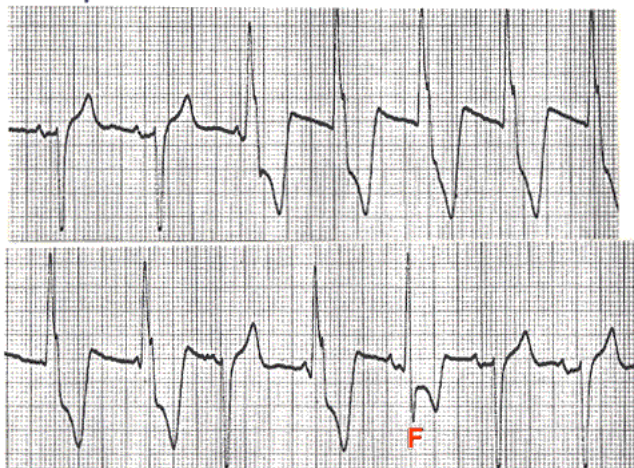


Incomplete AV dissociation due to sinus slowing (default) with junctional escapes (arrows)

[click here to view](#)

2. Acceleration of a subsidiary pacemaker faster than sinus rhythm; takeover by **usurpation**:

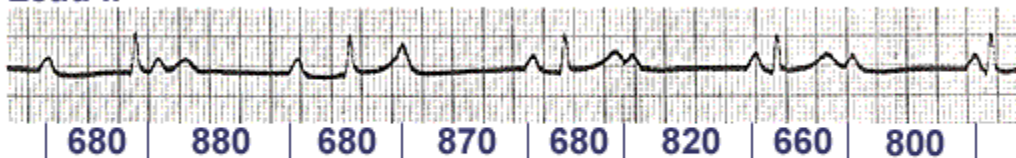
Lead V₁



Incomplete AV dissociation (usurpation) due to accelerated ventricular rhythm
F = fusion beat

3. 2nd or 3rd degree AV block with escape rhythm from junctional focus or ventricular focus:

Lead II



1. What is the diagnosis?

2. Why are the PP intervals alternating?

[click here to view](#)

In the above example of AV dissociation (3rd degree AV block with a junctional escape pacemaker) the PP intervals are alternating because of **ventriculophasic sinus arrhythmia** (phasic variation of vagal tone in the sinus node depending on the timing of ventricular contractions and blood flow near the carotid sinus).

4. Intraventricular Blocks

➤ **Right Bundle Branch Block (RBBB):**

● "Complete" RBBB has a QRS duration $\geq 0.12s$

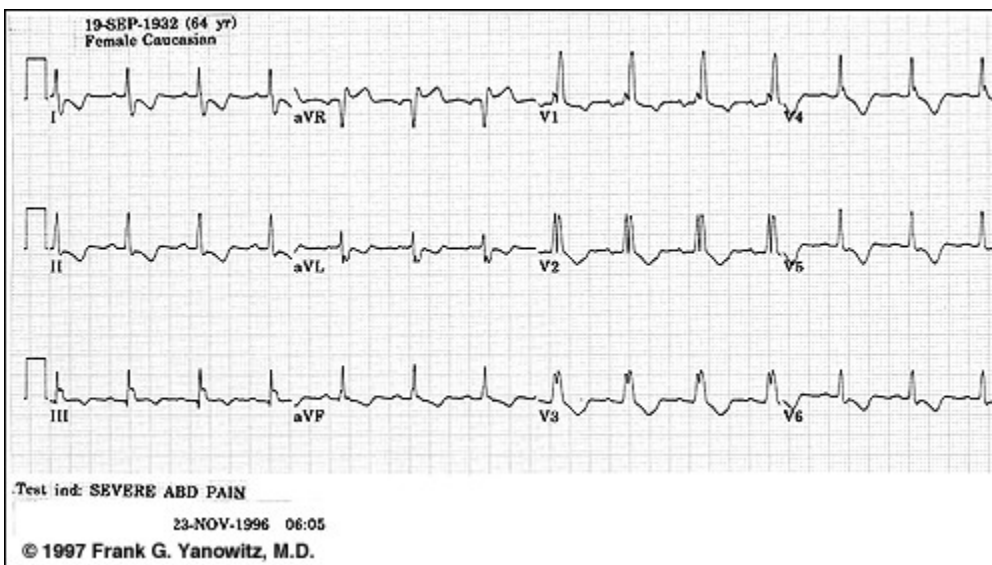
- Close examination of QRS complex in various leads reveals that the terminal forces (i.e., 2nd half of QRS) are oriented rightward and anteriorly because the right ventricle is depolarized **after** the left ventricle. This means the following:
 - Terminal R' wave in lead V1 (usually see rSR' complex) indicating late **anterior** forces
 - Terminal S waves in leads I, aVL, V6 indicating late **rightward** forces
 - Terminal R wave in lead aVR indicating late **rightward** forces

● The frontal plane QRS axis in RBBB should be in the normal range (i.e., -30 to +90 degrees). If left axis deviation is present, think about [left anterior fascicular block](#), and if right axis deviation is present, think about [left posterior fascicular block](#) in addition to the RBBB.

● "Incomplete" RBBB has a QRS duration of 0.10 - 0.12s with the same terminal QRS features. This is often a normal variant.

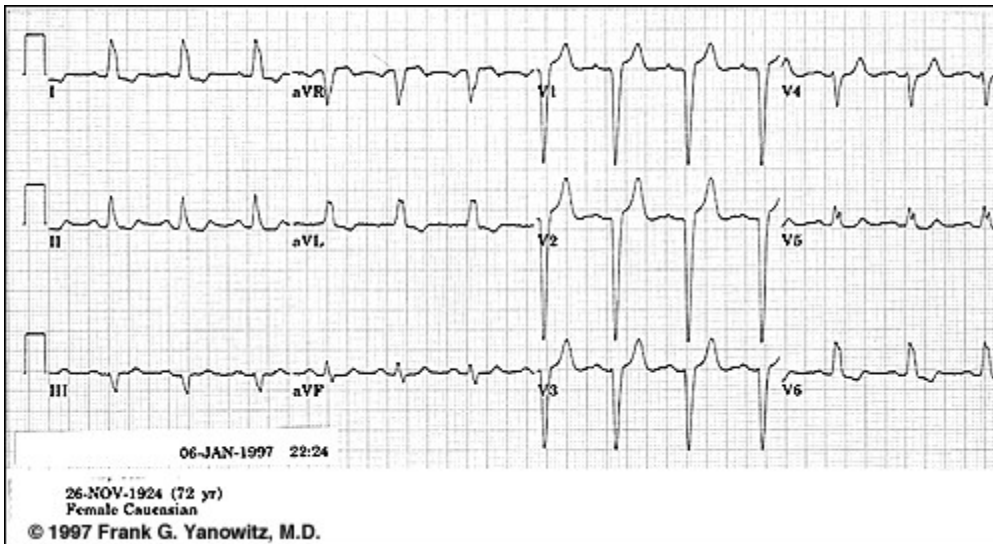
● The "normal" ST-T waves in RBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be negative or downwards; in leads with terminal S forces the ST-T should be positive or upwards. If the ST-T waves are in the **same direction** as the terminal QRS forces, they should be labeled **primary ST-T wave abnormalities**.

The ECG below illustrates primary ST-T wave abnormalities (leads I, II, aVR, V5, V6) in a patient with RBBB. ST-T wave abnormalities such as these may be related to ischemia, infarction, electrolyte abnormalities, medications, CNS disease, etc. (i.e., they are **nonspecific** and must be correlated with the patient's clinical status).



➤ Left Bundle Branch Block (LBBB)

- "Complete" LBBB has a QRS duration ≥ 0.12 s
- Close examination of QRS complex in various leads reveals that the terminal forces (i.e., 2nd half of QRS) are oriented leftward and posteriorly because the left ventricle is depolarized **after** the right ventricle.
 - Terminal S waves in lead V1 indicating late **posterior** forces
 - Terminal R waves in lead I, aVL, V6 indicating late **leftward** forces; usually broad, monophasic R waves are seen in these leads as illustrated in the ECG below; in addition, poor R progression from V1 to V3 is common.

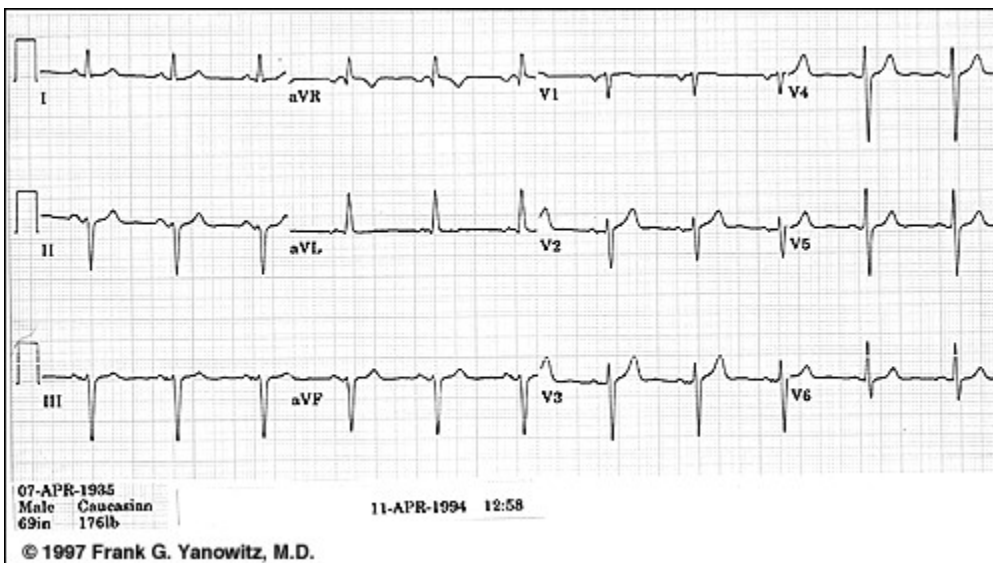


● The "normal" ST-T waves in LBBB should be oriented opposite to the direction of the terminal QRS forces; i.e., in leads with terminal R or R' forces the ST-T should be downwards; in leads with terminal S forces the ST-T should be upwards. If the ST-T waves are in the **same direction** as the terminal QRS forces, they should be labeled **primary ST-T wave abnormalities**. In the above ECG the ST-T waves are "normal" for LBBB; i.e., they are **secondary** to the change in the ventricular depolarization sequence.

● "Incomplete" LBBB looks like LBBB but QRS duration = 0.10 to 0.12s, with less ST-T change. This is often a progression of LVH.

➤ Left Anterior Fascicular Block (LAFB)... the most common intraventricular conduction defect

- Left axis deviation in frontal plane, usually -45 to -90 degrees
- rS complexes in leads II, III, aVF
- Small q-wave in leads I **and/or** aVL
- R-peak time in lead aVL >0.04s, often with slurred R wave downstroke
- QRS duration usually <0.12s unless coexisting RBBB
- Usually see poor R progression in leads V1-V3 and deeper S waves in leads V5 and V6
- May **mimic** LVH voltage in lead aVL, and **mask** LVH voltage in leads V5 and V6.



In this ECG, note -75 degree QRS axis, rS complexes in II, III, aVF, tiny q-wave in aVL, poor R progression V1-3, and late S waves in leads V5-6. QRS duration is normal, and there is a slight slur to the R wave downstroke in lead aVL.

➤ **Left Posterior Fascicular Block (LPFB)**.... Very rare intraventricular defect!

- Right axis deviation in the frontal plane (usually $> +100$ degrees)

- rS complex in lead I

- qR complexes in leads II, III, aVF, with R in lead III $>$ R in lead II

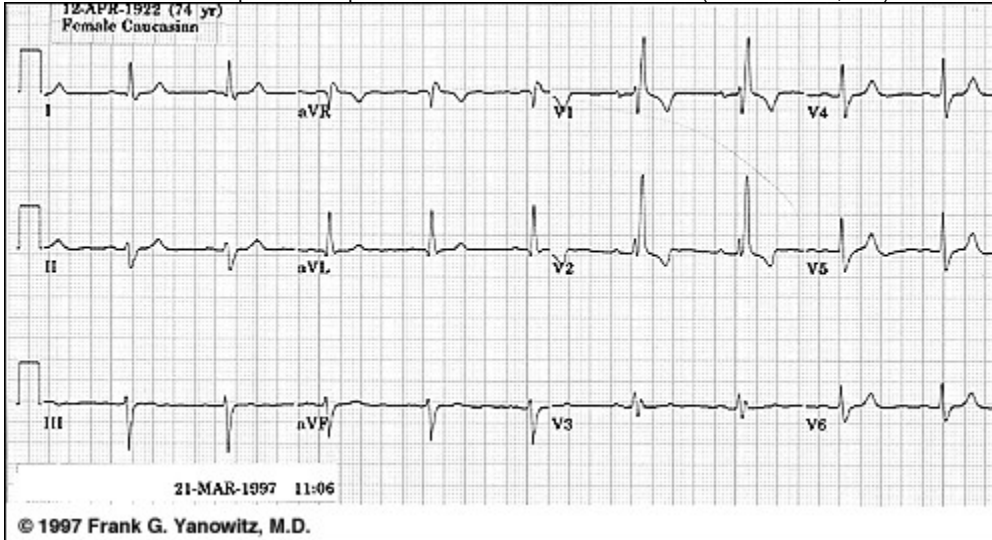
- QRS duration usually <0.12 s unless coexisting RBBB

- **Must first exclude (on clinical grounds) other causes of right axis deviation such as cor pulmonale, pulmonary heart disease, pulmonary hypertension, etc., because these conditions can result in the identical ECG picture!**

➤ **Bifascicular Blocks**

- RBBB plus either LAFB (common) **or** LPFB (uncommon)

- Features of RBBB plus frontal plane features of the fascicular block (axis deviation, etc.)



The above ECG shows classic RBBB (note rSR' in V1) **plus** LAFB (note QRS axis = -45 degrees, rS in II, III, aVF; and small q in aVL).

➤ **Nonspecific Intraventricular Conduction Defects (IVCD)**

- QRS duration >0.10 s indicating slowed conduction in the ventricles

- Criteria for specific bundle branch or fascicular blocks not met

- Causes of nonspecific IVCD's include:

- Ventricular hypertrophy (especially LVH)

- Myocardial infarction (so called **periinfarction blocks**)

- Drugs, especially class IA and IC antiarrhythmics (e.g., quinidine, flecainide)

- Hyperkalemia

➤ **Wolff-Parkinson-White Preexcitation**

- Although not a true IVCD, this condition causes widening of QRS complex and, therefore, deserves to be considered here

- QRS complex represents a **fusion** between **two** ventricular activation fronts:

- Early ventricular activation in region of the accessory AV pathway (**Bundle of Kent**)

- Ventricular activation through the normal AV junction, bundle branch system

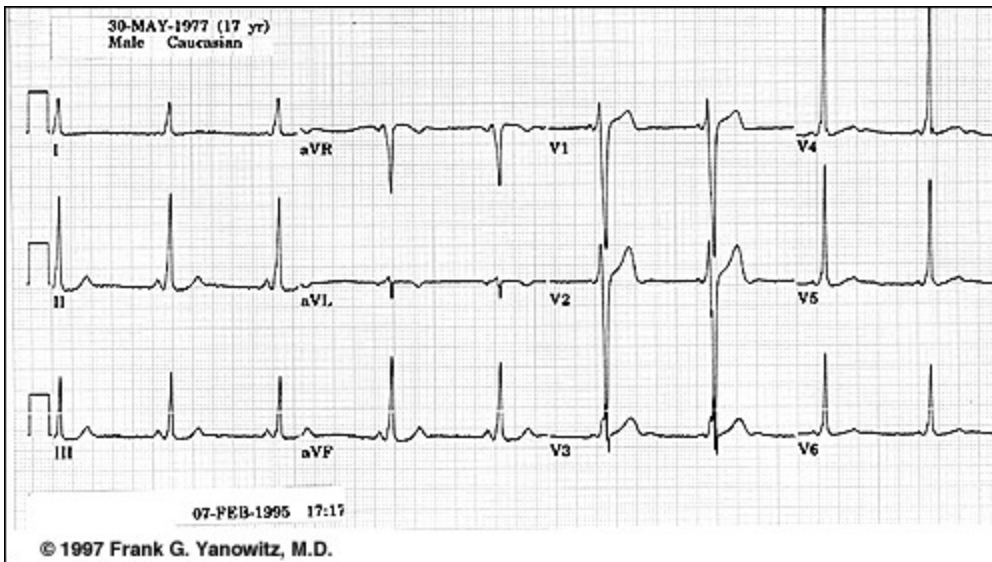
- ECG criteria include all of the following:

Short PR interval (<0.12 s)

Initial slurring of QRS complex (**delta wave**) representing early ventricular activation through normal ventricular muscle in region of the accessory pathway

Prolonged QRS duration (usually >0.10 s)

Secondary ST-T changes due to the altered ventricular activation sequence



● QRS morphology, including polarity of delta wave depends on the particular location of the accessory pathway as well as on the relative proportion of the QRS complex that is due to early ventricular activation (i.e., degree of fusion).

● Delta waves, if negative in polarity, may mimic infarct Q waves and result in false positive diagnosis of myocardial infarction.

VII. Atrial Enlargement

Topics for study:

[Right Atrial Enlargement \(RAE\)](#)

[Left Atrial Enlargement \(LAE\)](#)

[Bi-Atrial Enlargement \(BAE\)](#)

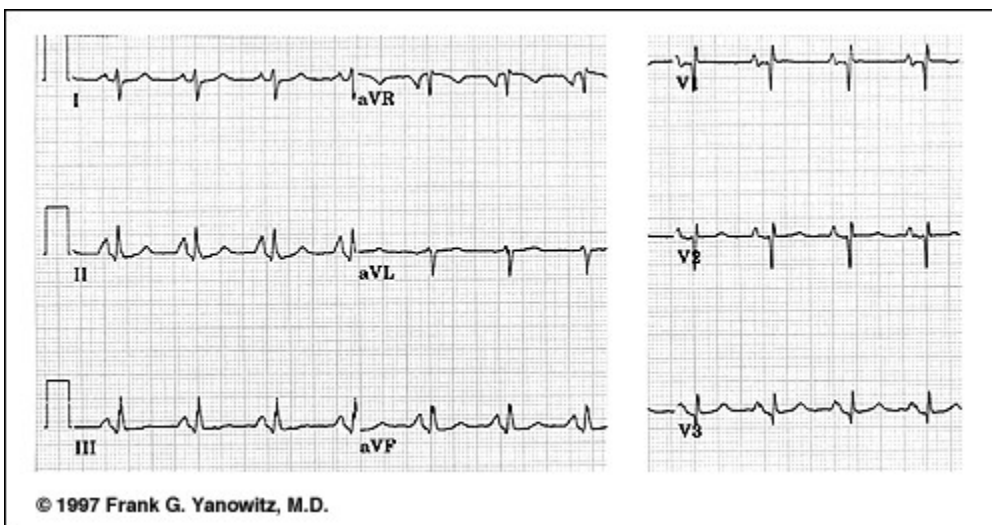
1. Right Atrial Enlargement (RAE)

➤ P wave amplitude >2.5 mm in II and/or >1.5 mm in V1 (these criteria are not very specific or sensitive)

➤ Better criteria can be derived from the QRS complex; these QRS changes are due to both the high incidence of RVH when RAE is present, and the RV displacement by an enlarged right atrium.

● QR, Qr, qR, or qRs morphology in lead V1 (in absence of coronary heart disease)

● QRS voltage in V1 is <5 mm and V2/V1 voltage ratio is >6 (Sensitivity = 50%; Specificity = 90%)

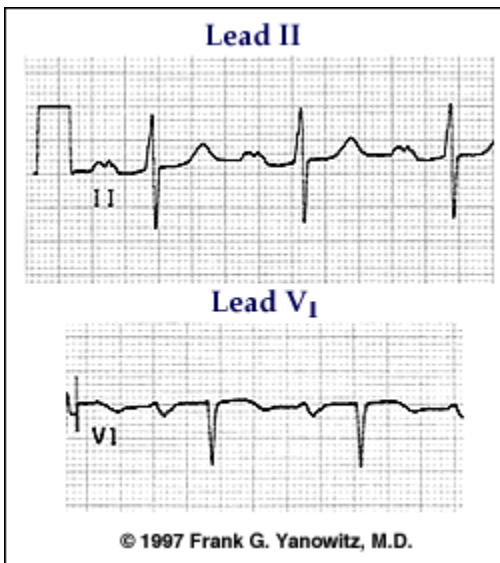


[click here to view](#)

In the above ECG, note the tall P waves in Lead II, and the Qr wave in Lead V1.

2. Left Atrial Enlargement (LAE)

- P wave duration ≥ 0.12 s in frontal plane (usually lead II)
 - Notched P wave in limb leads with the inter-peak duration ≥ 0.04 s
- Terminal P negativity in lead V1 (i.e., "P-terminal force") duration ≥ 0.04 s, depth ≥ 1 mm.
- Sensitivity = 50%; Specificity = 90%



3. Bi-Atrial Enlargement (BAE)

- Features of both RAE and LAE in same ECG
- P wave in lead II > 2.5 mm tall and ≥ 0.12 s in duration
- Initial positive component of P wave in V1 > 1.5 mm tall and prominent P-terminal force

VIII. Ventricular Hypertrophy

Topics for study:

[Introduction](#)

[Left Ventricular Hypertrophy \(LVH\)](#)

[Right Ventricular Hypertrophy \(RVH\)](#)

[Biventricular Hypertrophy](#)

1. Introductory Information:

- The ECG criteria for diagnosing right or left ventricular hypertrophy are very insensitive (i.e., sensitivity ~50%, which means that ~50% of patients with ventricular hypertrophy cannot be recognized by ECG criteria). However, the criteria are very specific (i.e., specificity >90%, which means if the criteria are met, it is very likely that ventricular hypertrophy is present).

2. Left Ventricular Hypertrophy (LVH)

- General ECG features include:
 - \geq QRS amplitude (voltage criteria; i.e., tall R-waves in LV leads, deep S-waves in RV leads)
 - Delayed intrinsicoid deflection in V6 (i.e., time from QRS onset to peak R is ≥ 0.05 sec)
 - Widened QRS/T angle (i.e., *left ventricular strain pattern*, or ST-T oriented opposite to QRS direction)
 - Leftward shift in frontal plane QRS axis
 - Evidence for [left atrial enlargement \(LAE\)](#) ([lesson VII](#))

➤ **ESTES** Criteria for LVH ("diagnostic", ≥5 points; "probable", 4 points)

+ECG Criteria	Points
Voltage Criteria (any of): R or S in limb leads ≥20 mm S in V1 or V2 ≥ 30 mm R in V5 or V6 ≥30 mm	3 points
ST-T Abnormalities: Without digitalis With digitalis	3 points 1 point
Left Atrial Enlargement in V1	3 points
Left axis deviation	2 points
QRS duration 0.09 sec	1 point
Delayed intrinsicoid deflection in V5 or V6 (>0.05 sec)	1 point

➤ **CORNELL** Voltage Criteria for LVH (sensitivity = 22%, specificity = 95%)

● S in V3 + R in aVL > 24 mm (men)

● S in V3 + R in aVL > 20 mm (women)

➤ Other Voltage Criteria for LVH

● Limb-lead voltage criteria:

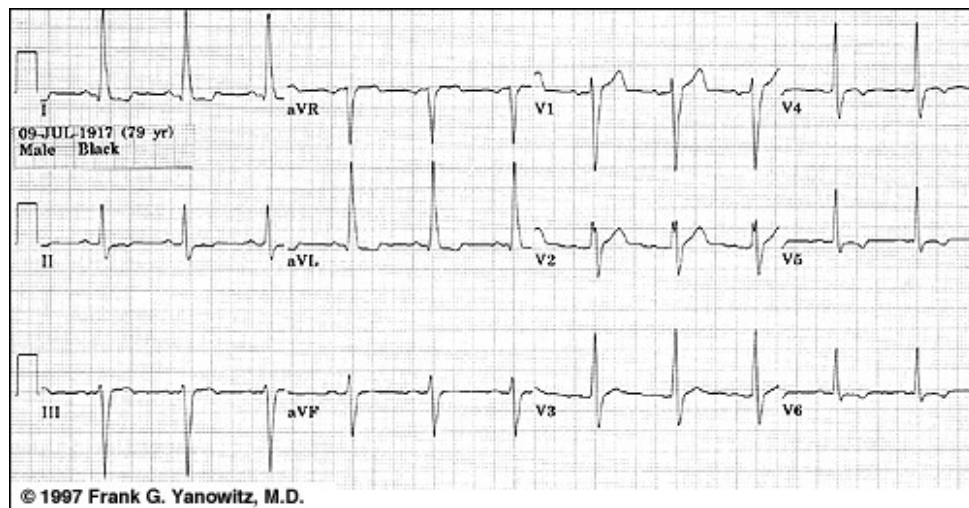
■ R in aVL ≥11 mm or, if left axis deviation, R in aVL ≥13 mm plus S in III ≥15 mm

■ R in I + S in III >25 mm

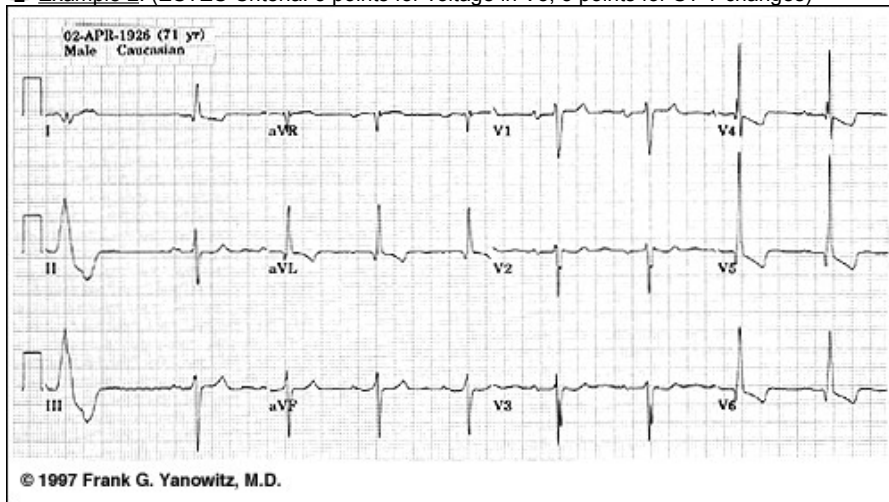
● Chest-lead voltage criteria:

■ S in V1 + R in V5 or V6 ≥ 35 mm

● Example 1: (Limb-lead Voltage Criteria; e.g., R in aVL >11 mm; note wide QRS/T angle)



● Example 2: (ESTES Criteria: 3 points for voltage in V5, 3 points for ST-T changes)



(Note also the left axis deviation of -40 degrees, and left atrial enlargement)

3. Right Ventricular Hypertrophy

➤ General ECG features include:

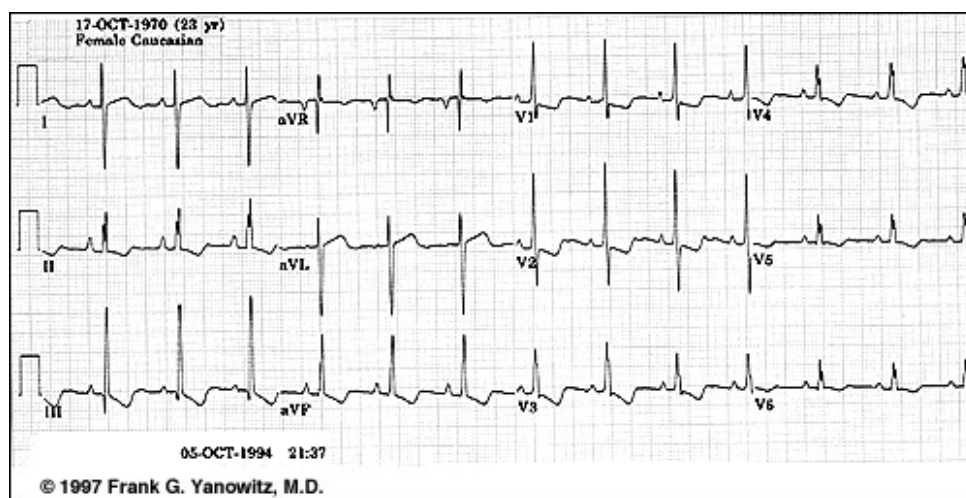
- Right axis deviation (>90 degrees)
- Tall R-waves in RV leads; deep S-waves in LV leads
- Slight increase in QRS duration
- ST-T changes directed opposite to QRS direction (i.e., wide QRS/T angle)
- May see incomplete RBBB pattern or qR pattern in V1
- Evidence of [right atrial enlargement \(RAE\)](#) ([lesson VII](#))

➤ Specific ECG features (assumes normal calibration of 1 mV = 10 mm):

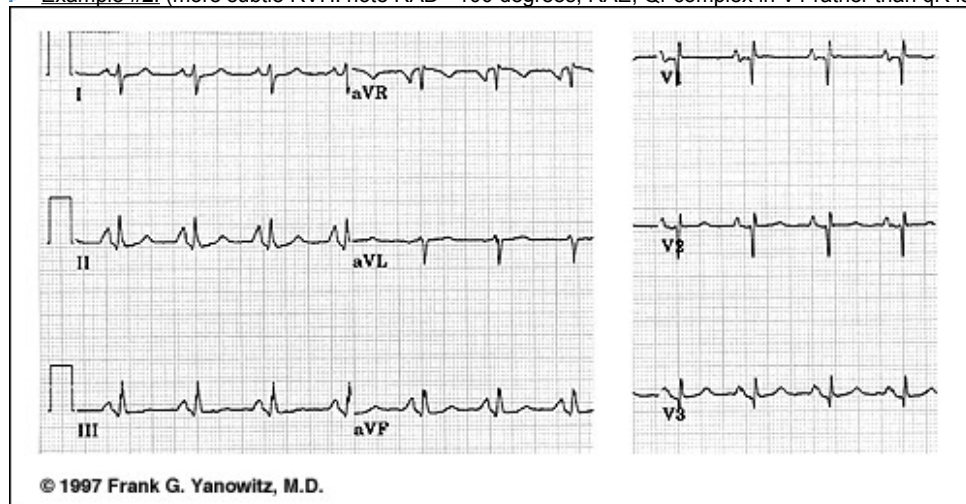
- Any one or more of the following (if QRS duration < 0.12 sec):
 - Right axis deviation (>90 degrees) in presence of disease capable of causing RVH
 - R in aVR \geq 5 mm, or
 - R in aVR > Q in aVR
- Any one of the following in lead V1:
 - R/S ratio > 1 and negative T wave
 - qR pattern
 - R > 6 mm, or S < 2mm, or rSR' with R' > 10 mm
- Other chest lead criteria:
 - R in V1 + S in V5 (or V6) 10 mm
 - R/S ratio in V5 or V6 < 1
 - R in V5 or V6 < 5 mm
 - S in V5 or V6 > 7 mm

● ST segment depression and T wave inversion in right precordial leads is usually seen in severe RVH such as in pulmonary stenosis and pulmonary hypertension.

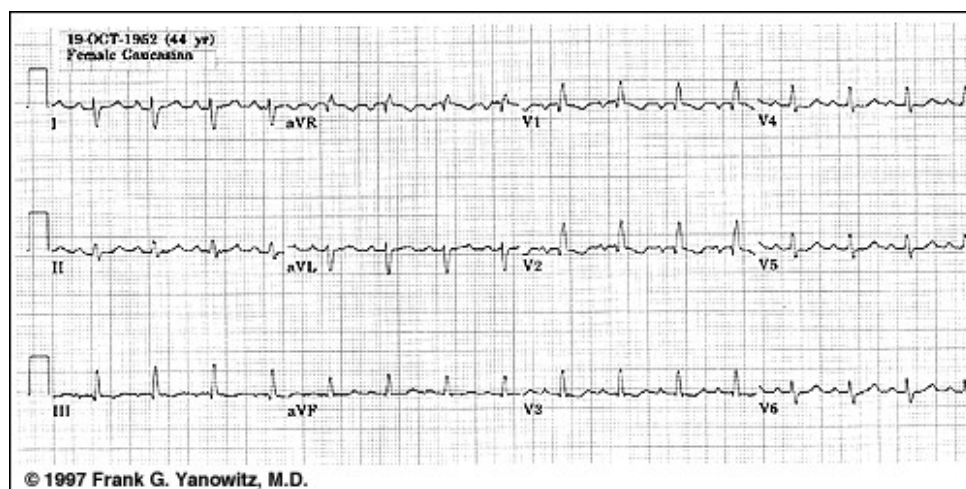
➤ Example #1: (note RAD +105 degrees; RAE; R in V1 > 6 mm; R in aVR > 5 mm)



➤ Example #2: (more subtle RVH: note RAD +100 degrees; RAE; Qr complex in V1 rather than qR is atypical)



➤ Example #3: (note: RAD +120 degrees, qR in V1; R/S ratio in V6 <1)



4. Biventricular Hypertrophy (difficult ECG diagnosis to make)

➤ In the presence of LAE any one of the following suggests this diagnosis:

● R/S ratio in V5 or V6 < 1

● S in V5 or V6 > 6 mm

● RAD (>90 degrees)

➤ Other suggestive ECG findings:

● Criteria for LVH and RVH both met

● LVH criteria met and RAD or RAE present

IX. Myocardial Infarction

Topics for study:

[Introduction](#) (Read this first)

[Inferior Q-Wave MI Family](#)

[Anterior Q-Wave MI Family](#)

[MI + Bundle Branch Block](#)

[Non Q-Wave MI](#)

[The Pseudoinfarctions](#)

[Miscellaneous QRS Abnormalities](#)

1. Introduction to ECG Recognition of Myocardial Infarction

➤ When myocardial blood supply is abruptly reduced or cut off to a region of the heart, a sequence of injurious events occur beginning with subendocardial or transmural ischemia, followed by necrosis, and eventual fibrosis (scarring) if the blood supply isn't restored in an appropriate period of time. Rupture of an atherosclerotic plaque followed by acute coronary thrombosis is the usual mechanism of acute MI. The ECG changes reflecting this sequence usually follow a well-known pattern depending on the location and size of the MI. MI's resulting from total coronary occlusion result in more homogeneous tissue damage and are usually reflected by a **Q-wave MI pattern** on the ECG. MI's resulting from subtotal occlusion result in more heterogeneous damage, which may be evidenced by a [non Q-wave MI](#) pattern on the ECG. **Two-thirds of MI's presenting to emergency rooms evolve to non-Q wave MI's, most having ST segment depression or T wave inversion.**

➤ Most MI's are located in the **left ventricle**. In the setting of a proximal right coronary artery occlusion, however, up to 50% may also have a component of [right ventricular infarction](#) as well. Right-sided chest leads are necessary to recognize RV MI.

➤ In general, the more leads of the 12-lead ECG with MI changes (Q waves and ST elevation), the larger the infarct size and the worse the prognosis. Additional leads on the back, V7-9 (horizontal to V6), may be used to improve the recognition of true posterior MI.

➤ The left anterior descending coronary artery (LAD) and its branches usually supply the anterior and anterolateral walls of the left ventricle and the anterior two-thirds of the septum. The left circumflex coronary artery (LCX) and its branches usually supply the posterolateral wall of the left ventricle. The right coronary artery (RCA) supplies the right ventricle, the inferior (diaphragmatic) and true posterior walls of the left ventricle, and the posterior third of the septum. The RCA also gives off the AV nodal coronary artery in 85-90% of individuals; in the remaining 10-15%, this artery is a branch of the LCX.

➤ Usual ECG evolution of a Q-wave MI; not all of the following patterns may be seen; the time from onset of MI to the final pattern is quite variable and related to the size of MI, the rapidity of reperfusion (if any), and the location of the MI.

● A. Normal ECG prior to MI

● B. Hyperacute T wave changes - increased T wave amplitude and width; may also see ST elevation

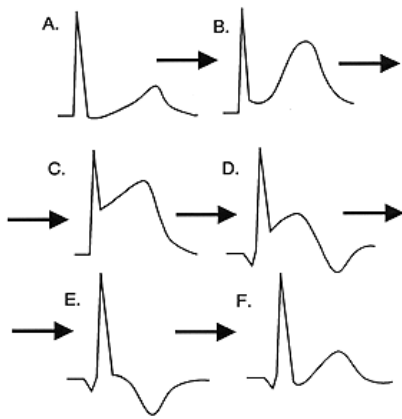
● C. Marked ST elevation with hyperacute T wave changes (transmural injury)

● D. Pathologic Q waves, less ST elevation, terminal T wave inversion (necrosis)

■ (Pathologic Q waves are usually defined as duration ≥ 0.04 s or >25% of R-wave amplitude)

● E. Pathologic Q waves, T wave inversion (necrosis and fibrosis)

● F. Pathologic Q waves, upright T waves (fibrosis)



Evolution of Acute MI

2. Inferior MI Family of Q-wave MI's

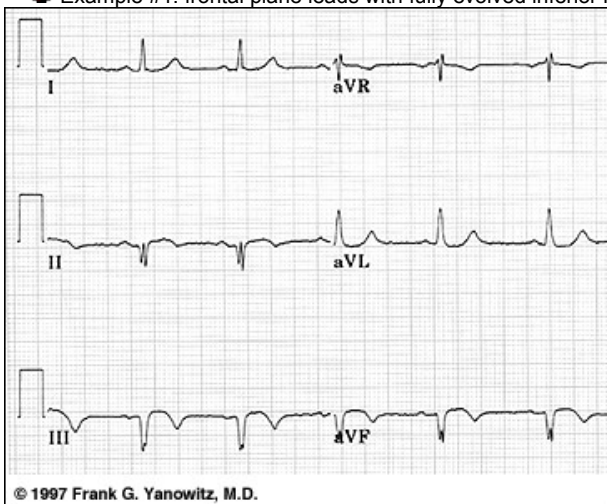
(includes inferior, true posterior, and right ventricular MI's)

➤ Inferior MI

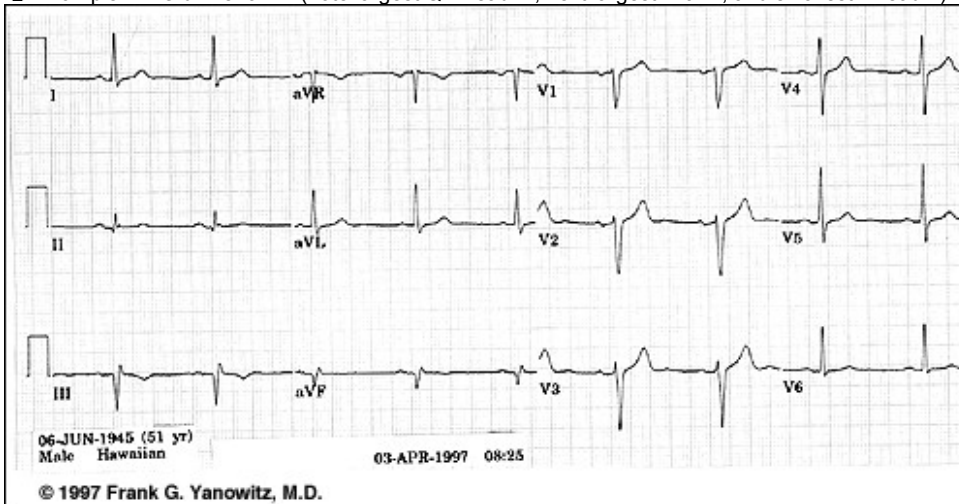
● Pathologic Q waves and evolving ST-T changes in leads II, III, aVF

● Q waves usually largest in lead III, next largest in lead aVF, and smallest in lead II

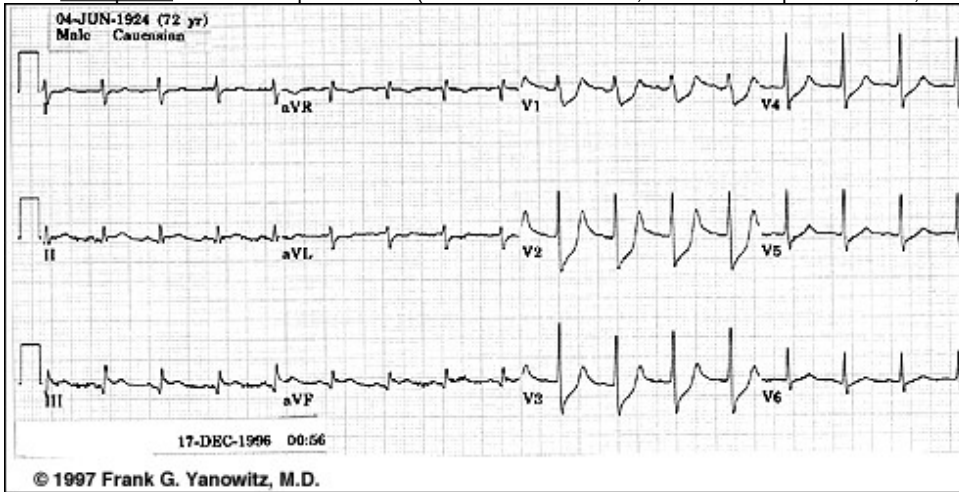
● Example #1: frontal plane leads with fully evolved inferior MI (note Q-waves, residual ST elevation, and T inversion in II, III, aVF)



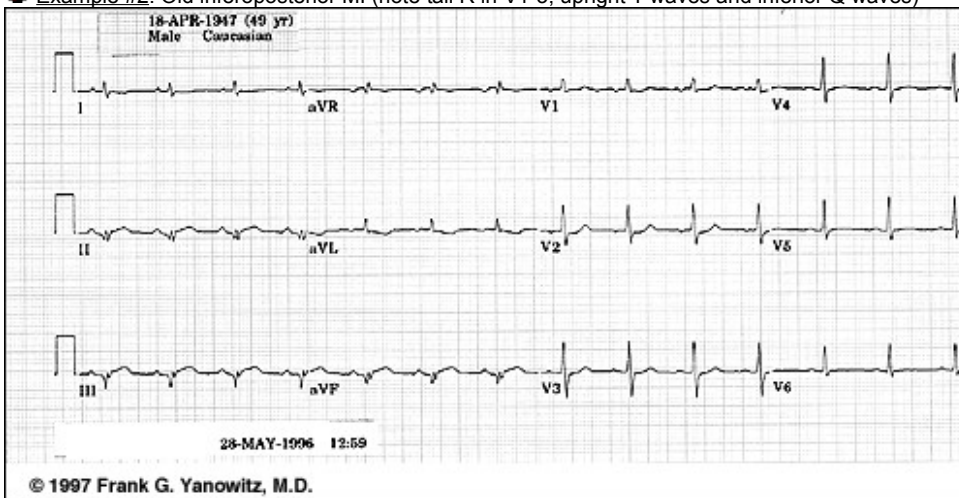
● Example #2: Old inferior MI (note largest Q in lead III, next largest in aVF, and smallest in lead II)



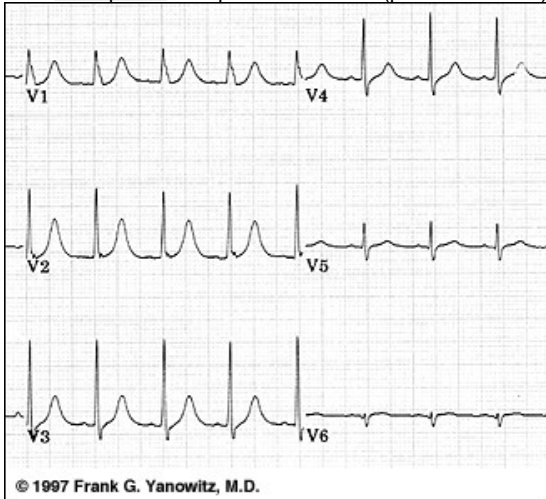
- True posterior MI
- ECG changes are seen in anterior precordial leads V1-3, but are the mirror image of an anteroseptal MI:
 - Increased R wave amplitude and duration (i.e., a "pathologic R wave" is a mirror image of a pathologic Q)
 - R/S ratio in V1 or V2 >1 (i.e., prominent anterior forces)
 - Hyperacute ST-T wave changes: i.e., ST depression and large, inverted T waves in V1-3
 - Late normalization of ST-T with symmetrical upright T waves in V1-3
- Often seen with inferior MI (i.e., "inferoposterior MI")
- **Example #1: Acute inferoposterior MI (note tall R waves V1-3, marked ST depression V1-3, ST elevation in II, III, aVF)**



- **Example #2: Old inferoposterior MI (note tall R in V1-3, upright T waves and inferior Q waves)**

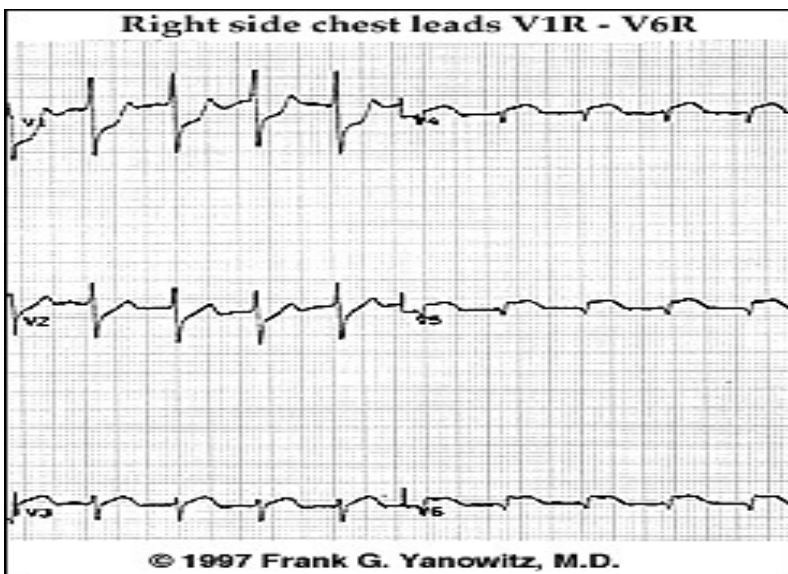


● **Example #3:** Old posterolateral MI (precordial leads): note tall R waves and upright T's in V1-3, and loss of R in V6



➤ Right Ventricular MI (only seen with proximal right coronary occlusion; i.e., with inferior family MI's)
 ● ECG findings usually require additional leads on right chest (V1R to V6R, analogous to the left chest leads)

● ST elevation, $\geq 1\text{mm}$, in right chest leads, especially V4R (see below)



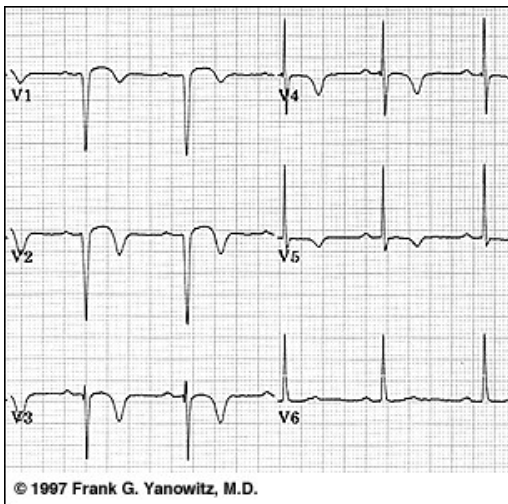
3. Anterior Family of Q-wave MI's

➤ Anteroseptal MI

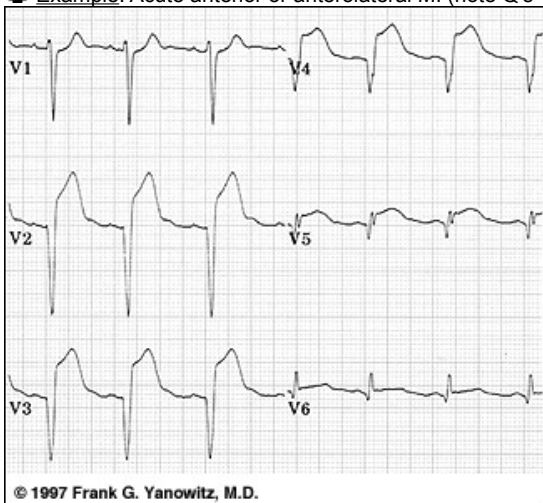
● Q, QS, or qrS complexes in leads V1-V3 (V4)

● Evolving ST-T changes

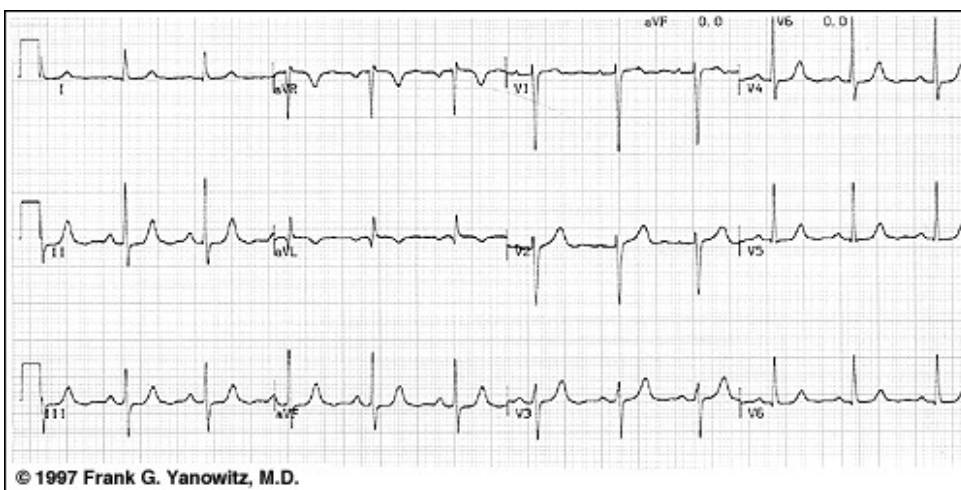
● **Example:** Fully evolved anteroseptal MI (note QS waves in V1-2, qrS complex in V3, plus ST-T wave changes)



- Anterior MI (similar changes, but usually V1 is spared; if V4-6 involved call it "anterolateral")
- **Example:** Acute anterior or anterolateral MI (note Q's V2-6 plus hyperacute ST-T changes)



- High Lateral MI (typical MI features seen in leads I and/or aVL)
- **Example:** note Q-wave, slight ST elevation, and T inversion in lead aVL



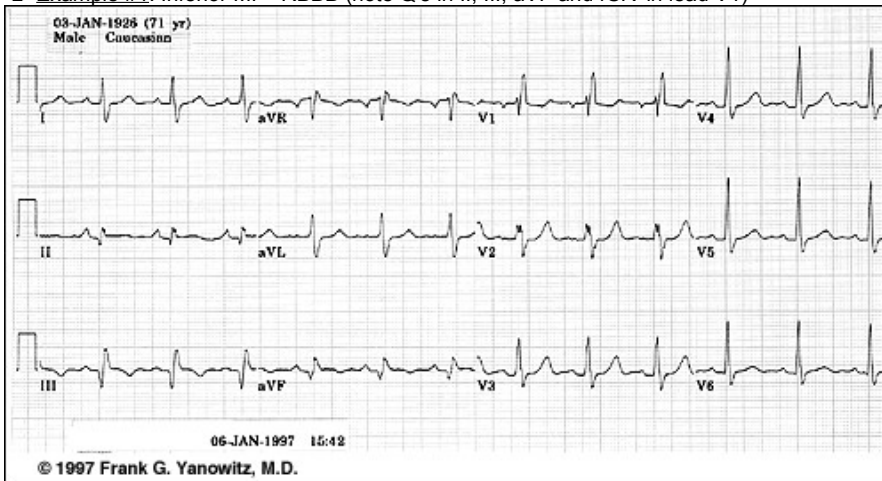
(Note also the slight U-wave inversion in leads II, III, aVF, V4-6, a strong marker for coronary disease)

4. MI with Bundle Branch Block

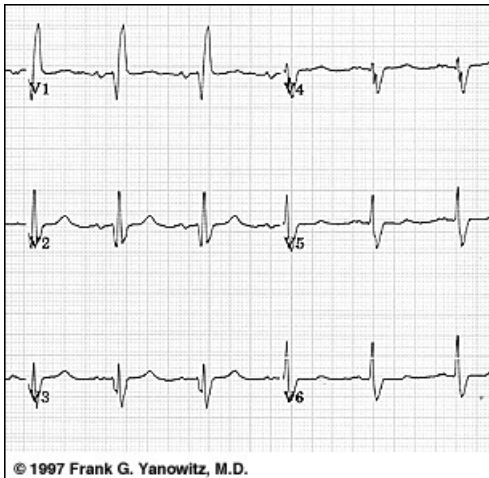
➤ MI + Right Bundle Branch Block

● Usually easy to recognize because Q waves and ST-T changes are not altered by the RBBB

● Example #1: Inferior MI + RBBB (note Q's in II, III, aVF and rSR' in lead V1)



● Example #2: Anteroseptal MI with RBBB (note Q's in leads V1-V3, terminal R wave in V1, fat S wave in V6)

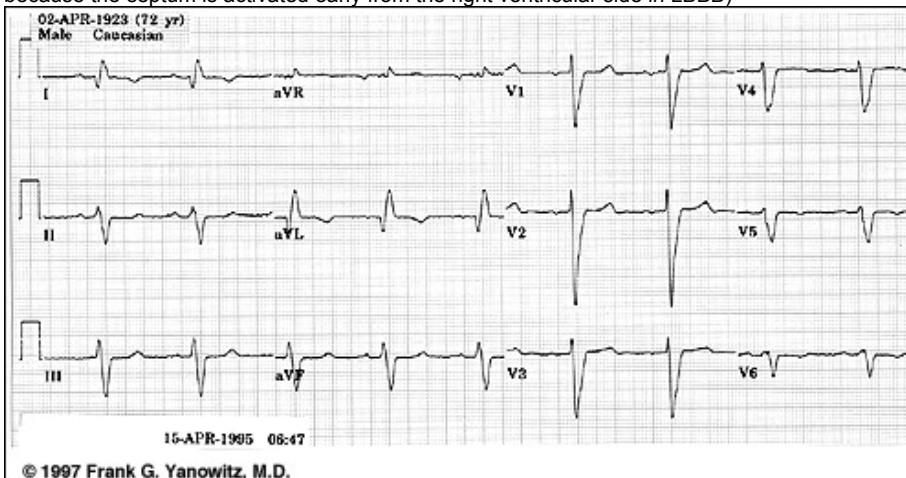


➤ MI + Left Bundle Branch Block

● Often a difficult ECG diagnosis because in LBBB the right ventricle is activated first and left ventricular infarct Q waves may not appear at the beginning of the QRS complex (unless the septum is involved).

● Suggested ECG features, not all of which are specific for MI include:

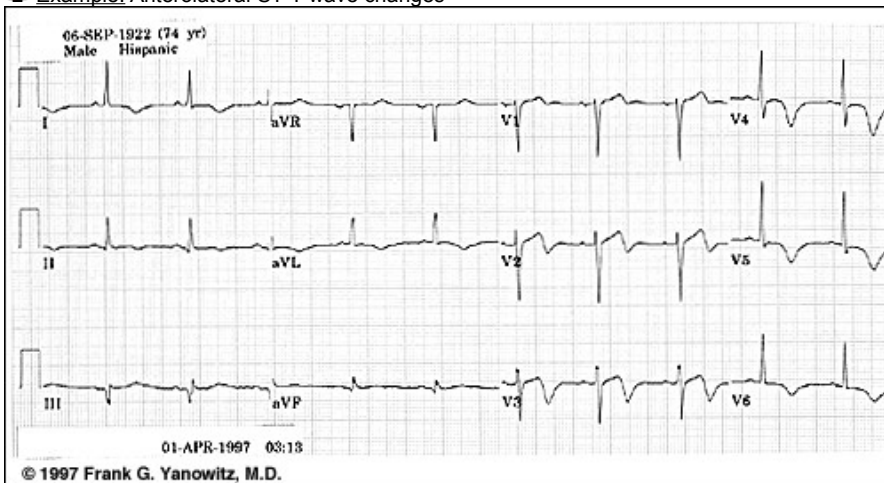
■ Q waves of any size in two or more of leads I, aVL, V5, or V6 (See below: one of the most reliable signs and probably indicates septal infarction, because the septum is activated early from the right ventricular side in LBBB)



- Reversal of the usual R wave progression in precordial leads (see above)
- Notching of the downstroke of the S wave in precordial leads to the right of the transition zone (i.e., before QRS changes from a predominate S wave complex to a predominate R wave complex); this may be a Q-wave equivalent.
- Notching of the upstroke of the S wave in precordial leads to the right of the transition zone (another Q-wave equivalent).
- rSR' complex in leads I, V5 or V6 (the S is a Q-wave equivalent occurring in the middle of the QRS complex)
- RS complex in V5-6 rather than the usual monophasic R waves seen in uncomplicated LBBB; (the S is a Q-wave equivalent).
- "Primary" ST-T wave changes (i.e., ST-T changes in the same direction as the QRS complex rather than the usual "secondary" ST-T changes seen in uncomplicated LBBB); these changes may reflect an acute, evolving MI.

5. Non-Q Wave MI

- Recognized by evolving ST-T changes over time without the formation of pathologic Q waves (in a patient with typical chest pain symptoms and/or elevation in myocardial-specific enzymes)
- Although it is tempting to localize the non-Q MI by the particular leads showing ST-T changes, this is probably only valid for the ST segment elevation pattern
- Evolving ST-T changes may include any of the following patterns:
 - Convex downward ST segment depression only (common)
 - Convex upwards or straight ST segment elevation only (uncommon)
 - Symmetrical T wave inversion only (common)
 - Combinations of above changes
- Example: Anterolateral ST-T wave changes



6. The Pseudoinfarcts

- These are ECG conditions that mimic myocardial infarction either by simulating pathologic Q or QS waves or mimicking the typical ST-T changes of acute MI.
- WPW preexcitation (*negative* delta wave may mimic pathologic Q waves)
- IHSS (septal hypertrophy may make normal septal Q waves "fatter" thereby mimicking pathologic Q waves)
- LVH (may have QS pattern or poor R wave progression in leads V1-3)
- RVH (tall R waves in V1 or V2 may mimic true posterior MI)
- Complete or incomplete LBBB (QS waves or poor R wave progression in leads V1-3)
- Pneumothorax (loss of right precordial R waves)
- Pulmonary emphysema and cor pulmonale (loss of R waves V1-3 and/or inferior Q waves with right axis deviation)
- Left anterior fascicular block (may see small q-waves in anterior chest leads)

- Acute pericarditis (the ST segment elevation may mimic acute transmural injury)
- Central nervous system disease (may mimic non-Q wave MI by causing diffuse ST-T wave changes)

7. Miscellaneous Abnormalities of the QRS Complex:

- The differential diagnosis of these QRS abnormalities depend on other ECG findings as well as clinical patient information
- Poor R Wave Progression - defined as loss of, or no R waves in leads V1-3 (R ≥ 2 mm):
 - Normal variant (if the rest of the ECG is normal)
 - LVH (look for voltage criteria and ST-T changes of LV "strain")
 - Complete or incomplete LBBB (increased QRS duration)
 - Left anterior fascicular block (should see LAD in frontal plane)
 - [Anterior or anteroseptal MI](#)
 - Emphysema and COPD (look for R/S ratio in V5-6 < 1)
 - Diffuse infiltrative or myopathic processes
 - WPW preexcitation (look for *delta waves*, short PR)
- Prominent Anterior Forces - defined as R/S ratio > 1 in V1 or V2
 - Normal variant (if rest of the ECG is normal)
 - [True posterior MI](#) (look for evidence of inferior MI)
 - RVH (should see RAD in frontal plane and/or P-pulmonale)
 - Complete or incomplete RBBB (look for rSR' in V1)
 - WPW preexcitation (look for *delta waves*, short PR)

X. ST Segment Abnormalities

Topics for study:

[General Introduction to ST-T and U Wave Abnormalities](#)
[ST Segment Elevation](#)
[ST Segment Depression](#)

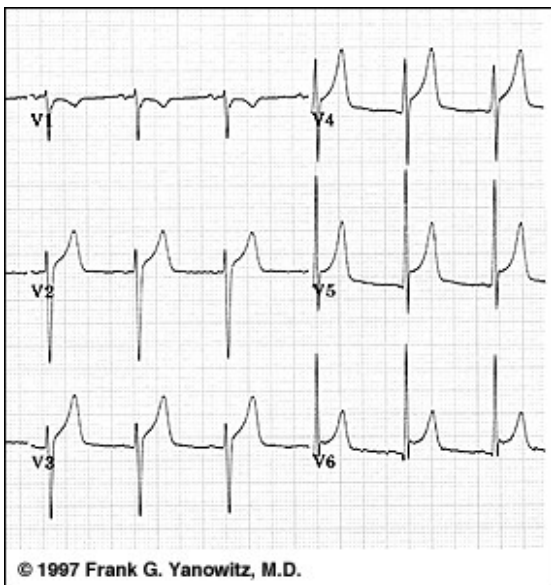
1. General Introduction to ST, T, and U wave abnormalities

- **Basic Concept:** the *specificity* of ST-T and U wave abnormalities is provided more by the clinical circumstances in which the ECG changes are found than by the particular changes themselves. Thus the term, *nonspecific ST-T wave abnormalities*, is frequently used when the clinical data are not available to correlate with the ECG findings. This does not mean that the ECG changes are unimportant! It is the responsibility of the clinician providing care for the patient to ascertain the importance of the ECG findings.
- Factors affecting the ST-T and U wave configuration include:
 - Intrinsic myocardial disease (e.g., myocarditis, ischemia, infarction, infiltrative or myopathic processes)
 - Drugs (e.g., digoxin, quinidine, tricyclics, and many others)
 - Electrolyte abnormalities of potassium, magnesium, calcium
 - Neurogenic factors (e.g., stroke, hemorrhage, trauma, tumor, etc.)
 - Metabolic factors (e.g., hypoglycemia, hyperventilation)
 - Atrial repolarization (e.g., at fast heart rates the atrial T wave may pull down the beginning of the ST segment)
 - Ventricular conduction abnormalities and rhythms originating in the ventricles

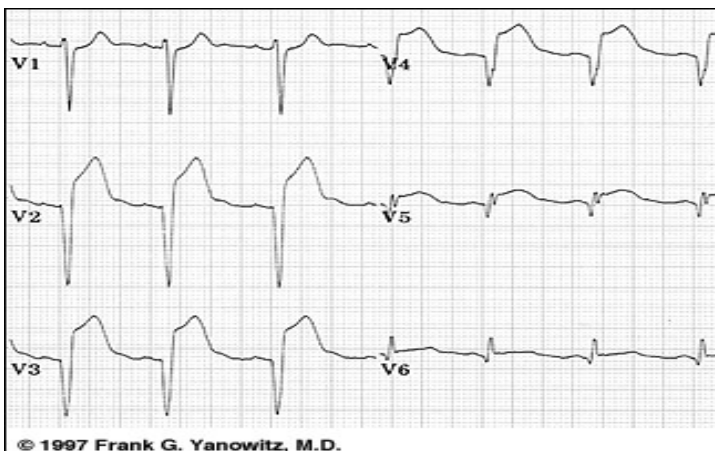
- "Secondary" ST-T Wave changes (these are normal ST-T wave changes solely due to alterations in the sequence of ventricular activation)
 - ST-T changes seen in bundle branch blocks (generally the ST-T polarity is opposite to the major or terminal deflection of the QRS)
 - ST-T changes seen in fascicular block
 - ST-T changes seen in nonspecific IVCD
 - ST-T changes seen in WPW preexcitation
 - ST-T changes in PVCs, ventricular arrhythmias, and ventricular paced beats
- "Primary" ST-T Wave Abnormalities (ST-T wave changes that are independent of changes in ventricular activation and that may be the result of global or segmental pathologic processes that affect ventricular repolarization)
 - Drug effects (e.g., digoxin, quinidine, etc)
 - Electrolyte abnormalities (e.g., hypokalemia)
 - Ischemia, infarction, inflammation, etc
 - Neurogenic effects (e.g., subarachnoid hemorrhage causing long QT)

2. Differential Diagnosis of ST Segment Elevation

- Normal Variant "Early Repolarization" (usually concave upwards, ending with symmetrical, large, upright T waves)
- Example #1: "Early Repolarization": note high take off of the ST segment in leads V4-6; the ST elevation in V2-3 is generally seen in most normal ECG's; the ST elevation in V2-6 is concave upwards, another characteristic of this normal variant.



- Ischemic Heart Disease (usually convex upwards, or straightened)
- Acute transmural injury - as in this acute anterior MI



[click here to view](#)

- Persistent ST elevation after acute MI suggests ventricular aneurysm
- ST elevation may also be seen as a manifestation of Prinzmetal's (variant) angina (coronary artery spasm)
- ST elevation during exercise testing suggests extremely tight coronary artery stenosis or spasm (transmural ischemia)

➤ Acute Pericarditis

- Concave upwards ST elevation in most leads except aVR
- No reciprocal ST segment depression (except in aVR)
- Unlike "early repolarization", T waves are usually low amplitude, and heart rate is usually increased.
- May see PR segment depression, a manifestation of atrial injury

➤ Other Causes:

- Left ventricular hypertrophy (in right precordial leads with large S-waves)
- Left bundle branch block (in right precordial leads with large S-waves)
- Advanced hyperkalemia
- Hypothermia (prominent J-waves or Osborne waves)

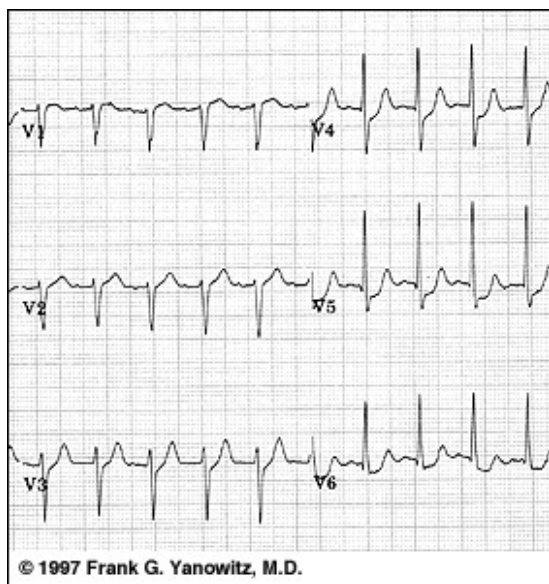
3. Differential Diagnosis of ST Segment Depression

➤ Normal variants or artifacts:

- Pseudo-ST-depression (wandering baseline due to poor skin-electrode contact)
- Physiologic J-junctional depression with sinus tachycardia (most likely due to atrial repolarization)
- Hyperventilation-induced ST segment depression

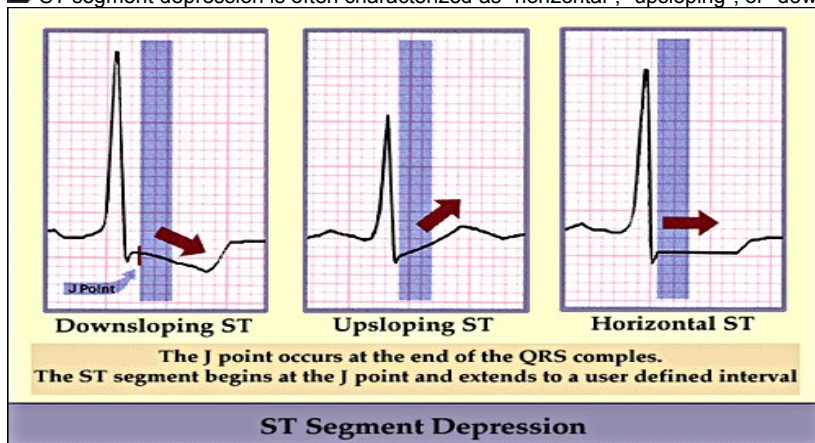
➤ Ischemic heart disease

- Subendocardial ischemia (exercise induced or during angina attack - as illustrated below)



Note: "horizontal" ST depression in lead V6

ST segment depression is often characterized as "horizontal", "upsloping", or "downsloping"



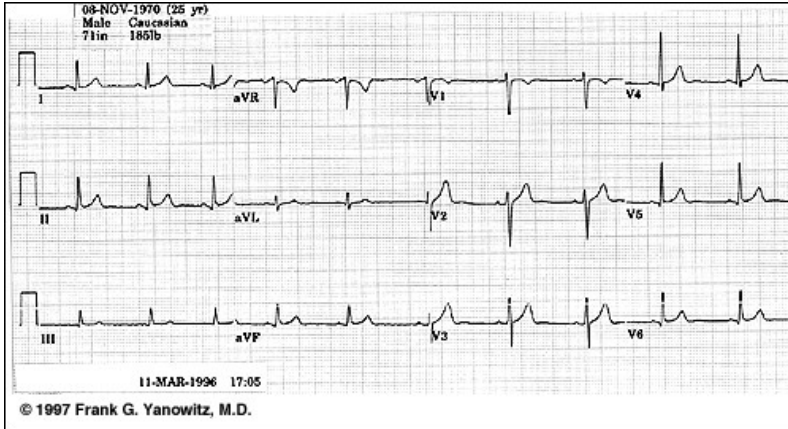
Note: "Upsloping" ST depression is not an ischemic abnormality

- Non Q-wave MI
- Reciprocal changes in acute Q-wave MI (e.g., ST depression in leads I & aVL with acute inferior MI)
- Nonischemic causes of ST depression
 - RVH (right precordial leads) or LVH (left precordial leads, I, aVL)
 - Digoxin effect on ECG
 - Hypokalemia
 - Mitral valve prolapse (some cases)
 - CNS disease
 - Secondary ST segment changes with IV conduction abnormalities (e.g., RBBB, LBBB, WPW, etc)

XI. T Wave Abnormalities

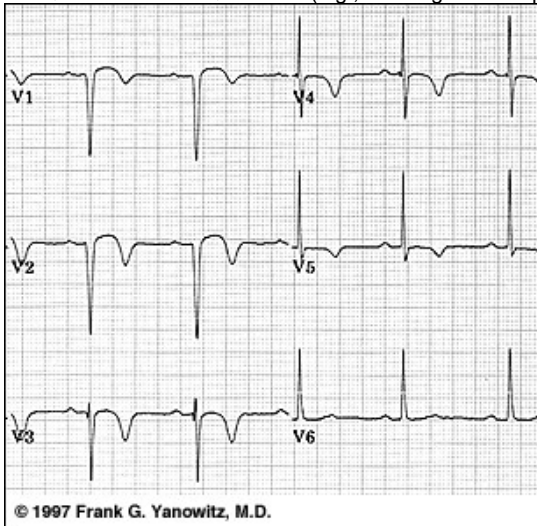
INTRODUCTION:

The T wave is the most labile wave in the ECG. T wave changes including **low-amplitude T waves** and **abnormally inverted T waves** may be the result of many cardiac and non-cardiac conditions. The normal T wave is usually in the same direction as the QRS except in the right precordial leads (see V2 below). Also, the normal T wave is *asymmetric* with the first half moving more slowly than the second half. In the normal ECG (see below) the T wave is always upright in leads I, II, V3-6, and always inverted in lead aVR. The other leads are variable depending on the direction of the QRS and the age of the patient.

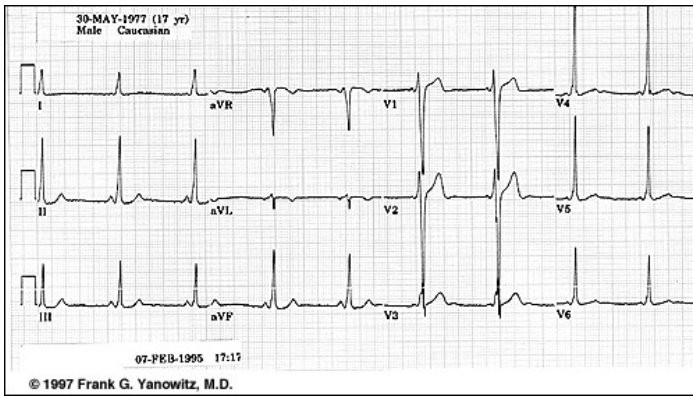


Differential Diagnosis of T Wave Inversion

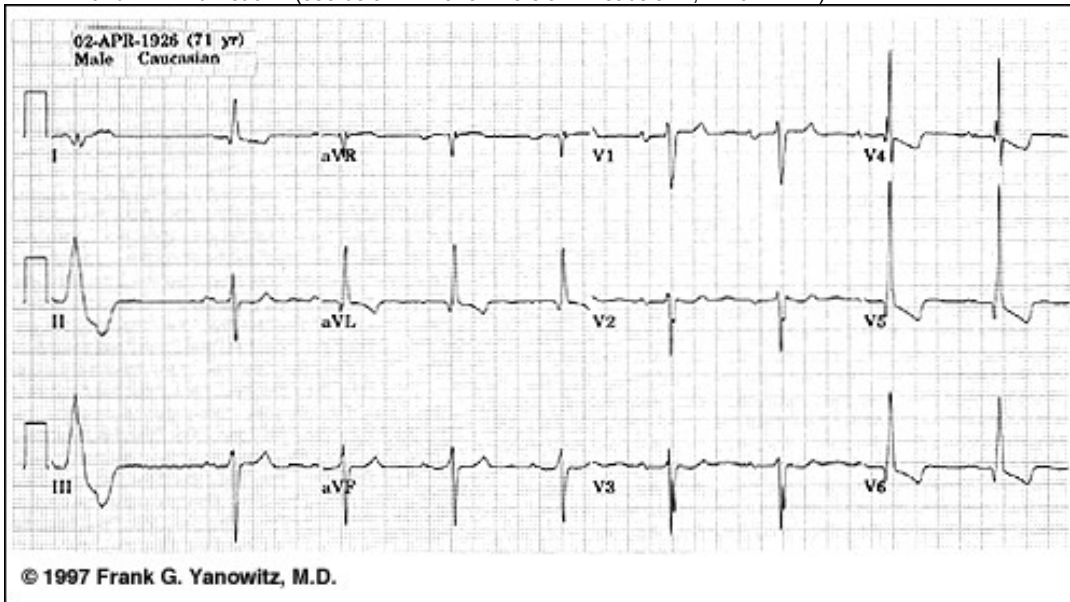
➤ Q wave and non-Q wave MI (e.g., evolving anteroseptal MI):



- Myocardial ischemia
- Subacute or old pericarditis
- Myocarditis
- Myocardial contusion (from trauma)
- CNS disease causing long QT interval (especially subarachnoid hemorrhage; see below):



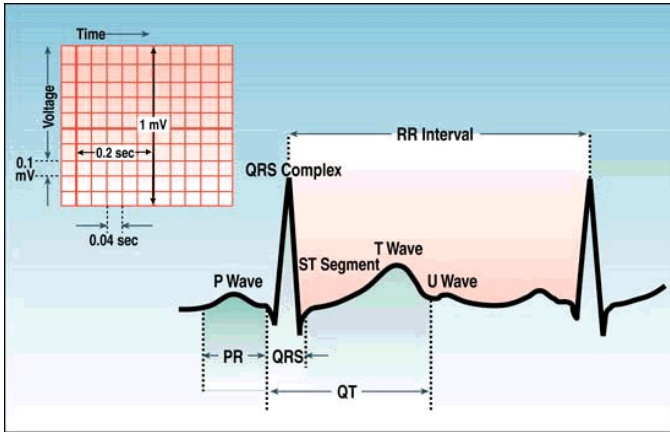
- Idiopathic apical hypertrophy (a rare form of hypertrophic cardiomyopathy)
- Mitral valve prolapse
- Digoxin effect
- RVH and LVH with "strain" (see below: T wave inversion in leads aVL, V4-6 in LVH)



XII. Nice Seeing "U" Again

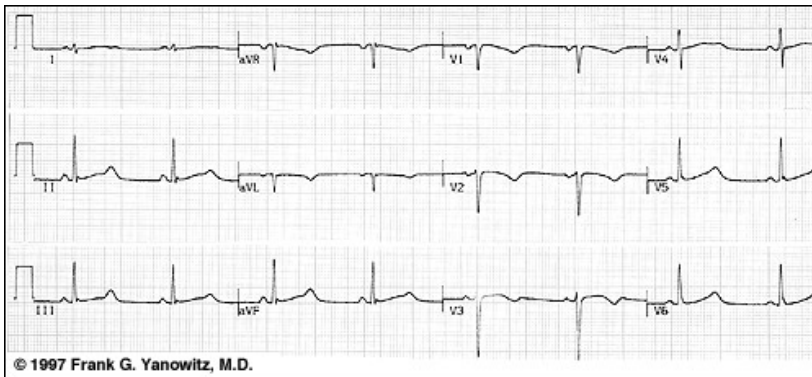
Introduction:

The U wave is the only remaining enigma of the ECG, and probably not for long. The origin of the U wave is still in question, although most authorities correlate the U wave with electrophysiologic events called "afterdepolarizations" in the ventricles. These afterdepolarizations can be the source of arrhythmias caused by "triggered automaticity" including *torsade de pointes*. The normal U wave has the same polarity as the T wave and is usually less than one-third the amplitude of the T wave. U waves are usually best seen in the right precordial leads especially V2 and V3. The normal U wave is asymmetric with the ascending limb moving more rapidly than the descending limb (just the opposite of the normal T wave).



Differential Diagnosis of U Wave Abnormalities

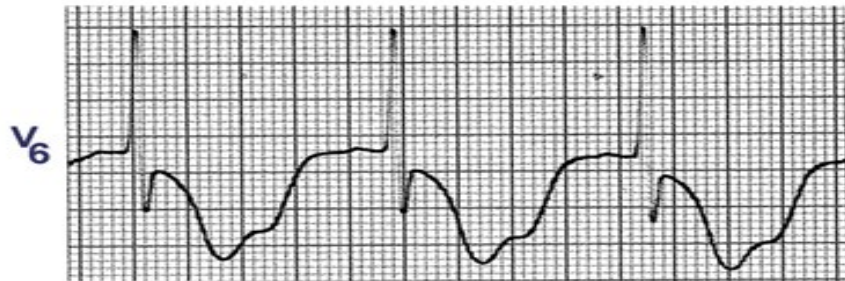
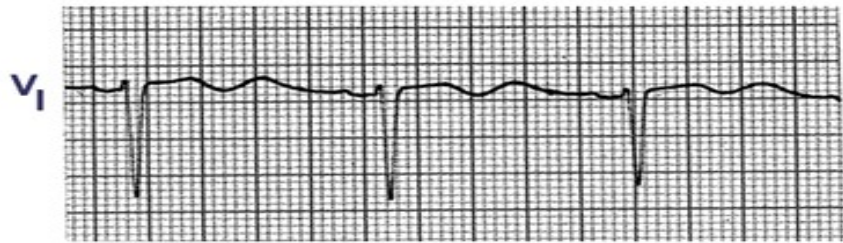
- Prominent upright U waves
- Sinus bradycardia accentuates the U wave
- Hypokalemia (remember the triad of ST segment depression, low amplitude T waves, and prominent U waves)
- Quinidine and other type 1A antiarrhythmics
- CNS disease with long QT intervals (often the T and U fuse to form a giant "T-U fusion wave")



(E.g., lead II, III, V4-6)

- LVH (right precordial leads with deep S waves)
- Mitral valve prolapse (some cases)
- Hyperthyroidism
- Negative or "inverted" U waves
- Ischemic heart disease (often indicating left main or LAD disease)
- Myocardial infarction (in leads with pathologic Q waves)
- During episode of acute ischemia (angina or exercise-induced ischemia)
- Post extrasystolic in patients with coronary heart disease
- During coronary artery spasm (Prinzmetal's angina)

- Nonischemic causes
- Some cases of LVH or RVH (usually in leads with prominent R waves)
- Some patients with LQTS (see below: Lead V6 shows giant negative TU fusion wave in patient with LQTS; a prominent upright U wave is seen in Lead V1)



'U'-nbelievable